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ABSTRACT

This TIP on the best practice guidelines for treatment of substance abuse provides basic knowledge for practitioners, educators, and paraprofessionals about the nature and treatment of stimulant use disorders. More specifically, it reviews what is currently known about treating the medical, psychiatric, and substance abuse/dependence problems associated with the use of cocaine and methamphetamine. It includes a discussion on how stimulants affect the brain and behavior. It suggests that psychosocial treatment approaches that incorporate well established psychological principles of learning are appropriate for and effective in treating stimulant users. In addition, it discusses the community-reinforcement-plus-vouchers approach, the Matrix Model, and behavioral family therapy. Other models of psychosocial treatment discussed include network therapy, acupuncture, and inpatient treatment. Pharmacological treatments are also considered along with other medical aspects of stimulant use disorders. The information on treatment issues for special groups (i.e., intravenous drug users, gay men, individuals with co-occurring mental disorders) and settings (rural areas) underscores the need for cultural competence in the treatment setting. Appendixes include: "Bibliography," Client Worksheets," "Screening Tests for Cognitive Impairments," "Glossary," "Resource Panel," and "Field Reviewers." (Contains 31 figures and approximately 300 resources.) (JDM)

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Treatment for Stimulant Use Disorders

Treatment Improvement Protocol (TIP) Series

33

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CSAT *Treatment Improvement Protocol* (TIP) 33: Treatment for Stimulant Use Disorders

Treatment Improvement Protocols (TIPs) are best practice guidelines for the treatment of substance use disorders that are researched, drafted, and reviewed by substance use disorder professionals. The guidelines are developed during a week-long consensus panel, at which experts discuss a particular topic in the substance use disorder field. These discussions are the basis for the first draft of the TIP, which is reviewed by professionals nationwide.

Treatment for Stimulant Use Disorders, TIP 33, supplies treatment providers with vital information on the effects of stimulant abuse and dependence, discusses the relevance of these effects to treating stimulant users, describes treatment approaches that are appropriate and effective for treating these clients, and makes specific recommendations on the practical application of these treatment strategies. These scientifically validated strategies recognize the impact that stimulant use has on an individual's ability to respond to treatment and address the specific problems and needs that are inherent to chronic stimulant users. For clinicians and primary care providers, the document provides an overview of the medical aspects of stimulant use. In addition, this TIP reviews treatment issues for special groups and settings. Also included are over 40 client worksheets to assist providers in treating people with stimulant use disorders.

This is what the expert reviewers had to say about *Treatment for Stimulant Use Disorders*:

"An excellent document!"

"I found this to be a readable and well-prepared document that will be extremely helpful to the treatment community."

"This TIP is detailed and comprehensive."

"There is a good deal of information clearly provided in [this TIP] ... very clear and understandable."

"The treatment strategies and medical aspects were very informative."

"The client worksheets included in this TIP are most useful."

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Highlights of Recommendations

Treatment Approaches

- Psychosocial treatment approaches that incorporate well-established psychological principles of learning are most appropriate for treating stimulant abusers.
- A contingency management approach, particularly the community-reinforcement-plus-vouchers approach, has been shown to be most effective for these clients.

Maximizing Treatment Engagement

- Make treatment accessible.
- Provide support for treatment participation.
- Respond quickly and positively to initial telephone inquiries.

Assessment and Orientations

- Keep initial assessments brief.
- Provide clear orientations.
- Offer clients treatment options.
- Involve significant others.
- Convey empathetic concern.

Initiating Treatment

- Establish treatment attendance.
- Discontinue use of psychoactive substances and initiate urinalysis schedule.
- Assess psychiatric comorbidity.
- Assess stimulant-associated compulsive sexual behavior.
- Remediate stimulant "withdrawal" symptoms.
- Resolve any immediate crisis.

Initiating Abstinence

- Establish structure and support.
- Address secondary drug use.
- Initiate avoidance strategies.
- Provide client education.
- Respond to early slips.

Maintaining Abstinence

- Teach functional analysis of stimulant use.
- Teach relapse prevention techniques.
- Enhance self-efficacy regarding high-risk situations.
- Counteract euphoric recall and desire to test control.

Relapse Prevention

Successful relapse-prevention approaches systematically teach clients the following:

- How to cope with substance craving
- Substance refusal assertiveness skills
- How seemingly irrelevant decisions may affect the probability of later substance use
- General coping and problem-solving skills
- How to apply strategies to prevent a full-blown relapse should an episode of substance use occur

Treatment for Stimulant Use Disorders

Treatment Improvement Protocol (TIP) Series

33

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The opinions expressed herein are the views of the Consensus Panel members and do not reflect the official position of CSAT, SAMHSA, or the U.S. Department of Health and Human Services (DHHS). No official support or endorsement of CSAT, SAMHSA, or DHHS for these opinions or for particular instruments or software that may be described in this document is intended or should be inferred. The guidelines proffered in this document should not be considered as substitutes for individualized client care and treatment decisions.

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What Is a TIP?

Treatment Improvement Protocols (TIPs) are best practice guidelines for the treatment of substance use disorders, provided as a service of the Substance Abuse and Mental Health Services Administration's Center for Substance Abuse Treatment (CSAT). CSAT's Office of Evaluation, Scientific Analysis and Synthesis draws on the experience and knowledge of clinical, research, and administrative experts to produce the TIPs, which are distributed to a growing number of facilities and individuals across the country. The audience for the TIPs is expanding beyond public and private substance use disorder treatment facilities as alcoholism and other substance use disorders are increasingly recognized as major problems.

The TIPs Editorial Advisory Board, a distinguished group of substance use experts and professionals in such related fields as primary care, mental health, and social services, works with the State Alcohol and Other Drug Abuse Directors to generate topics for the TIPs based on the field's current needs for information and guidance.

After selecting a topic, CSAT invites staff from pertinent Federal agencies and national organizations to a Resource Panel that recommends specific areas of focus as well as resources that should be considered in developing the content of the TIP. Then recommendations are communicated to a Consensus Panel composed of non-Federal experts on the topic who have been nominated by their peers. This Panel participates in a series of discussions; the information and recommendations on which they reach consensus form the foundation of the TIP. The members of each Consensus Panel represent substance use disorder treatment programs, hospitals, community health centers, counseling programs, criminal justice and child welfare agencies, and private practitioners. A Panel Chair (or Co-Chairs) ensures that the guidelines mirror the results of the group's collaboration.

A large and diverse group of experts closely reviews the draft document. Once the changes recommended by these field reviewers have been incorporated, the TIP is prepared for publication, in print and online. The TIPs can be accessed via the Internet on the National Library of Medicine's home page at the URL: http://text.nlm.nih.gov. The move to electronic media also means that the TIPs can be updated more easily so they continue to provide the field with state-of-the-art information.

Although each TIP strives to include an evidence base for the practices it recommends, CSAT recognizes that the field of substance use disorder treatment is evolving and that research frequently lags behind the innovations pioneered in the field. A major goal of each TIP is to convey "front line" information quickly but responsibly. For this reason, recommendations proffered in the TIP are attributed to either Panelists' clinical experience or the literature. If there is research to support a particular approach, citations are provided.

This TIP, *Treatment for Stimulant Use Disorders*, supplies substance use disorder treatment providers with vital information on the effects of stimulant abuse and dependence, discusses the relevance of these effects to treating stimulant users, describes treatment approaches that are appropriate and effective for treating these clients, and makes specific recommendations on the practical application of these treatment strategies. Research on animals has demonstrated the profound effects that stimulants can have on the central nervous system, and new technologies have begun to document the stimulant-induced neurological impairments in humans. Researchers now believe that these impairments underlie the cognitive deficits that are often seen in chronic stimulant users.

Effective treatment strategies must recognize the impact that stimulant abuse and dependence have on the user's ability to respond to treatment. The treatment strategies that are described in this TIP have been scientifically validated as effective in treating people with stimulant use disorders. These strategies address the specific problems and needs that are inherent to chronic stimulant users.

This document discusses in detail the practical application of these treatment strategies and makes recommendations to improve treatment outcomes for stimulant abuse and dependence. The document also provides for clinicians and primary care providers an overview of the medical aspects of stimulant use. Furthermore, treatment issues for special groups and settings are reviewed.

This TIP represents another step by CSAT toward its goal of bringing national leaders together to improve substance use disorder treatment in the United States.

Other TIPs may be ordered by contacting SAMHSA's National Clearinghouse for Alcohol and Drug Information (NCADI), (800) 729-6686 or (301) 468-2600; TDD (for hearing impaired), (800) 487-4889.

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Foreword

The Treatment Improvement Protocol (TIP) series fulfills SAMHSA/CSAT's mission to improve treatment of substance use disorders by providing best practices guidance to clinicians, program administrators, and payors. TIPs are the result of careful consideration of all relevant clinical and health services research findings, demonstration experience, and implementation requirements. A panel of non-Federal clinical researchers, clinicians, program administrators, and client advocates debates and discusses their particular area of expertise until they reach a consensus on best practices. This panel's work is then reviewed and critiqued by field reviewers.

The talent, dedication, and hard work that TIPs panelists and reviewers bring to this highly participatory process have bridged the gap between the promise of research and the needs of practicing clinicians and administrators. We are grateful to all who have joined with us to contribute to advances in the substance use disorder treatment field.

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Executive Summary and Recommendations

Over the last 20 years, the use of stimulants has risen to national and international prominence. Stimulant use and its consequences have brought havoc to many communities across the United States and have prompted strong responses from Federal, State, and local governments and organizations. For example, the relatively minor problems of cocaine use in the 1960s and 1970s have grown to become major medical, legislative, and law enforcement issues in the 1990s. The devastation wrought by the crack cocaine epidemic is familiar to most Americans.

Similarly, the use and abuse of another stimulant, methamphetamine (MA), have risen dramatically in recent years. Widespread use and abuse of MA have led to a greater awareness of the problem and have inspired policymakers, legal officials, and service providers to focus increased efforts toward the personal and societal effects of this drug. Concerns that MA abuse may result in another epidemic led to passage of the Comprehensive Methamphetamine Control Act of 1996.

The explosive growth of stimulant use triggered a flurry of research. The results are tremendous advances in fundamental knowledge of stimulant use disorders and on the basic function of the brain and addictive disorders in general. Yet today, there are few reports that describe either the fundamentals of stimulant use disorder treatment or the success of various treatment interventions.

This Treatment Improvement Protocol (TIP) describes basic knowledge about the nature and treatment of stimulant use disorders. More specifically, it reviews what is currently known about treating the medical, psychiatric, and substance abuse/dependence problems associated with the use of two high-profile stimulants: cocaine and MA.

The scientifically based information in this TIP is presented in a manner that makes it available and relevant for clinicians and other "front line" substance use disorder treatment providers. It offers recommendations on treatment approaches, recommendations to maximize treatment engagement, strategies for planning and initiating treatment, and strategies for initiating and maintaining abstinence. Also included are recommendations for the medical management of stimulant users and recommendations regarding special groups and settings.

The Consensus Panel that developed this TIP tried to emphasize those treatment techniques and principles that have been established with empirical support. However, because the "science" of treating stimulant use disorders is barely a decade old, the Panel also reviewed and synthesized a set of techniques and principles developed and supported by leading addiction specialists, but with less empirical support. This document delineates those treatment suggestions and recommendations that are empirically supported and those that are currently based on consensus opinion.

The purpose of this TIP is to advance the understanding of treating the substance use disorders associated with the abuse of cocaine and MA. The Consensus Panel's recommendations summarized below are based on both researched and clinical experience. Those supported by scientific evidence are followed by (1); clinically based recommendations are marked (2). Citations to the former are referenced in the body of this document, where the guidelines are presented in full detail. To avoid sexism and awkward sentence construction, the TIP alternates between "he" and "she" in generic examples.

For purposes of this TIP, the substances included in the category of "stimulants" include the derivatives of the coca plant (cocaine hydrochloride and its freebase form, "crack") and the synthetically produced amphetamines, with a primary emphasis on illicitly produced MA (and its smokable form, "ice"). Certainly there are other stimulants that are more widely used (e.g., caffeine) or that produce major health problems (e.g., nicotine); however, an extensive discussion of issues associated with these substances is beyond the scope of this document.

Summary of Recommendations

Because of recent health care reforms, most individuals who seek help for stimulant dependence now receive treatment at structured outpatient treatment programs. Accordingly, this document provides recommendations for treatment strategies and techniques that are most relevant to the treatment of stimulant-dependent patients in structured outpatient treatment programs. However, many, if not most, of these strategies and techniques can be integrated into other types of programs, regardless of the setting or therapeutic orientation.

Psychosocial Treatment Approaches

Psychosocial treatment approaches that incorporate well established psychological principles of learning are appropriate for and effective in treating stimulant users. In an effort to make these approaches consistently effective, the Consensus Panel recommends the use of carefully prepared treatment manuals to minimize differences among therapists. (2) Treatment manuals increase the likelihood that therapists will deliver a uniform set of services to their clients. However, the therapist's clinical judgment and flexibility are extremely important to the treatment process.

The Consensus Panel recommends a contingency management approach for treating stimulant users. (1) A particularly successful version is the community-reinforcement-plus-vouchers approach in which couples counseling, vocational training, and skills training are combined with rewards for negative drug tests (i.e., "clean" urinalysis results).

Relapse Prevention

Relapse prevention systematically teaches clients

- How to cope with substance craving
- Substance refusal assertiveness skills
- How seemingly irrelevant decisions may affect the probability of later substance use
- General coping and problem-solving skills
- How to apply strategies to prevent a full-blown relapse should an episode of substance use occur

The Consensus Panel recommends this approach for use with stimulant users. (1)

Other Interventions With Supportive Research

Research indicates that the following may be appropriate interventions for stimulant users:

- Permitting women entering residential treatment to be accompanied by some or all of their children (1)
- Supportive–expressive psychotherapy (1)
- "Node-link mapping," which uses flowcharts and other methods to diagram relationships between clients' thoughts, actions, feelings, and substance use (1)

Other Models of Psychosocial Treatment

A number of other psychosocial models and approaches have been described, and some used widely, for the treatment of stimulant use disorders, including:

- Network therapy, in which clients receiving individual psychotherapy develop a network of stable, nonsubstance-abusing support persons, such as family, partners, and close friends (2)
- Acupuncture (2)
- Therapeutic communities (the most common type of long-term residential treatment) (1)

Maximizing Treatment Engagement

Make treatment accessible

To maximize treatment engagement, programs must make treatment accessible. Having treatment programs in areas convenient to clients is associated with lower attrition rates. (1) Treatment should be provided during the hours and on the days that are convenient for clients. (2) Programs should be located near public transportation and in a part of town viewed as safe for evening visits. (2)

Provide support for treatment participation

Address clients' concrete needs, including transportation, housing, and finances. (1) Some logistical barriers can be overcome by onsite services, through agreements with subcontractors, or by referrals. These can include onsite child care services, referrals to temporary shelters, vouchers for lunches, targeted financial assistance, assistance with paperwork regarding insurance, or filing for disability benefits. (2)

Respond quickly and positively to initial telephone inquiries

Because ambivalence about treatment is common among treatment-seeking stimulant users, methods to "screen out" those who are "in denial" are counterproductive and impede treatment entry. (2) The initial interview should be scheduled within 24 hours after the client initially contacts the program. (2)

Assessments and Orientations

Keep initial assessments brief

Initial assessments should be brief, focused, and nonrepetitive. (2)

Provide clear orientations

Individuals need a thorough, clear, and realistic orientation about stimulant use disorder treatment. Clients should acquire a good understanding about the treatment process, the rules of the treatment program, expectations about their participation, and what they can expect the program to do for them and in what time frame. (2)

Offer clients options

Addiction treatment is more effective when a client chooses it from among alternatives than when it is assigned as the only option. Thus, it is important to provide clients with options and negotiate with them regarding the treatment approaches and strategies that are the most acceptable and promising. (1)

Involve significant others

Whenever possible, family and significant others who support the treatment goals should be involved in the treatment process. (2)

Convey empathetic concern

Counselors should be warm, friendly, engaging, empathetic, straightforward, and nonjudgmental. Authoritarian and confrontational behavior by the staff can substantially increase the potential for violence. (2)

Planning Treatment

To organize treatment strategies, it can be helpful to view the treatment process as consisting of

- A treatment initiation period
- An abstinence attainment period
- An abstinence maintenance phase
- A long-term abstinence support plan

The Consensus Panel recommends treatment for 12 to 24 weeks followed by some type of support group participation. (2)

Clients should have a written schedule of expected attendance they can keep and give to family members who may be involved in treatment. It does not appear appropriate to deliver these services on an ad hoc or as needed basis. (2)

Initiating Treatment

The initial period of stimulant abstinence is characterized by symptoms of depression, difficulty concentrating, poor memory, irritability, fatigue, craving for cocaine/MA, and paranoia (especially for MA users). The duration of these symptoms varies; in general, symptoms typically last 3 to 5 days for cocaine users and 10 to 15 days for MA users. (2)

The first several weeks of treatment have some relatively simple and straightforward priorities.

Establish treatment attendance

During the first 2 or 3 weeks, clients should be scheduled for multiple weekly visits, even if the visits are 30 minutes in duration or less. (2)

Discontinue use of psychoactive substances and initiate urinalysis schedule

Immediately upon entering the treatment program, clients should be placed on a mandatory, vigilant, and frequent urine testing schedule. This schedule should continue throughout the treatment process, although the frequency of testing can be tapered as treatment progresses. Urine samples should be taken every 3 or 4 days so as not to exceed the sensitivity limits of standard laboratory testing methods. (2) Participation in self-help groups should be strongly encouraged but not required.

Assess psychiatric comorbidity

During the initial 2 weeks of treatment, it is important to assess the possible existence of other psychiatric conditions and, if present, initiate appropriate treatment, including medication. (2)

Assess stimulant-associated compulsive sexual behaviors

Research has revealed an association between stimulant use and a variety of compulsive sexual behaviors. These behaviors include promiscuous sex, AIDS-risky behaviors, compulsive masturbation, compulsive pornographic viewing, and homosexual behavior for otherwise heterosexual individuals. In order for treatment to be effective, these issues must be discussed openly and nonjudgmentally. (2)

Remediate stimulant "withdrawal" symptoms

Remind clients that proper sleep and nutrition are necessary to allow the neurobiology of the brain to "recover." Giving them "permission" to sleep, eat, and gradually begin a program of exercise, can help establish some behaviors that will have long-term utility. These behaviors will help them begin to think more clearly and begin to feel some benefit from their initial efforts in treatment. (1)

Initiating Abstinence

Establish structure and support. After the initial treatment engagement of 1 to 2 weeks, the focus is on the achievement of abstinence. Although there is no clear delineation between clients who are *initiating* abstinence and those *maintaining* abstinence, the initiating period occurs roughly from 2 to 6 weeks into treatment. (2)

Establish structure and support

Short-term goals should be set immediately and should be reasonably achievable. One such goal is complete abstinence from all substances for 1 week. (2)

Brief, frequent counseling sessions can reinforce the short-term goal of immediate abstinence and establish a therapeutic alliance between the client and counselor. Events of the past 24 hours are reviewed in each session and recommendations are provided for navigating the next 24 hours. (2)

Address secondary drug use

For many clients, their secondary substance use may not have been associated with adverse consequences or compulsive use. As a result, such clients need help to identify the connections between the use of other substances and their stimulant addiction. (2)

Clients should be encouraged to throw out all substance-related items. (2) Family members, sober friends, or 12-Step sponsors should help with this task.

Initiate avoidance strategies

Clients must develop specific action plans to break contacts with dealers and other stimulant users and to avoid high-risk places that are strongly associated with stimulant use. (2)

Provide client education

Educate clients about learning and conditioning factors associated with stimulant use and the impact of stimulants and other substances on the brain and behavior, such as cognitive impairments and forgetfulness. (2)

Other steps to initiate abstinence include

- Identify cues and triggers (2)
- Develop action plan for cues and triggers (2)
- Enlist family participation (2)
- Establish social support systems (2)
- Address stimulant abuse–associated compulsive sexual behaviors (2)

Respond to early slips

Early slips should not be considered tragic failures but rather simple mistakes. When slips occur, counselors can make a verbal or behavioral contract with clients regarding short-term achievable goals. (2)

Maintaining Abstinence

Teach functional analysis of stimulant use

The core components of a functional analysis are

- Teaching clients to examine the types of circumstances, situations, thoughts, and feelings that increase the likelihood that they will use stimulants
- Counseling clients to examine the positive, immediate, but short-term consequences of their stimulant use
- Encouraging clients to review the negative and often delayed consequences of their stimulant use (2)

Teach relapse prevention techniques

Relapse prevention techniques fall into the following categories:

- Psychoeducation about the relapse process and how to interrupt it
- Identification of high-risk situations and relapse warning signs
- Developing coping and stress management skills
- Enhancing self-efficacy in dealing with potential relapse situations
- Counteracting euphoric recall and the desire to test control over use

- Developing a balanced lifestyle including healthy leisure and recreation activities
- Responding safely to slips to avoid escalation into full-blown relapse
- Establishing behavioral accountability for slips and relapse via urine monitoring and/or Breathalyzer™ testing (2)

Enhance self-efficacy regarding high-risk situations

Once clients learn to identify, manage, and avoid high-risk situations, the counselor and client should try to determine if the client is confident in her ability to use those skills in the real world through role-playing and other therapeutic techniques. (2)

Counteract euphoric recall and desire to test control

So-called "war stories" that include euphoric recall and selective memory are powerful relapse triggers and should be strongly discouraged in recovery groups. (2)

Medical Aspects

The following recommendations are for medical personnel to help them recognize and treat problems that may arise for stimulant users with acute or chronic intoxication or in various phases of withdrawal.

The most common reasons for emergency room visits by cocaine users are cardiopulmonary symptoms (usually chest pains or palpitations); psychiatric complaints, ranging from altered mental states to suicidal ideation; and neurological problems, including seizures and delirium.

The major presenting symptoms for MA users pertain primarily to altered mental status, including confusion, delusions, paranoid reactions, hallucinations, and suicidal ideation. The rapid development of tolerance to its physiological effects among chronic MA users may explain the relative infrequency of cardiac complications in this group. (1)

The lethal dose of cocaine for 50 percent of novice users (LD_{50}) is 1.5 grams. The LD_{50} for MA has not specifically been established, and there is significant individual variability to its toxicity. For example, doses of 30 milligrams can produce severe reactions, yet doses of 400 to 500 milligrams are not necessarily fatal. (1)

Management of stimulant intoxication

Uncomplicated intoxication requires only observation and monitoring in a subdued environment until symptoms subside over several hours.

Physical exertion and an overheated room can potentiate adverse effects because stimulants affect the body's heat-regulating mechanism at the same time that blood vessel constriction conserves heat.

Indications that agitation is escalating and moving toward paranoia and potential psychosis (losing touch with reality), with increasing risk for violence, may warrant pharmacological intervention. Fast-acting benzodiazepines such as lorazepam (Ativan) or diazepam (Valium) are useful for calming an anxious, agitated client. (1)

Management of potentially lethal overdose

- Manage hyperthermia by sedating to slow down and stop agitated movements and by rapidly cooling the client with body ice packs, mist and fan techniques, or cooling blankets. (1)
- If restraints are required to start an intravenous administration, use mesh-type blankets only transiently to avoid interfering further with heat loss. (2)
- Uncontrolled hypertension can be managed by intravenous administration of phentolamine (Regitine) or dopamine (Intropin). (1)
- Treat seizures like status epilepticus with intravenous diazepam or other

2.,

benzodiazepine. Diazepam is most effective if administered before or shortly after cocaine ingestion but is less effective after seizures begin. (1)

Management of stimulant withdrawal

The greatest risk from the distinctive stimulant abstinence syndrome is that one may do harm to oneself or others. Because withdrawal-related dysphoria and depression can be particularly severe in stimulant users, risk of suicide is intensified, and sensitive management is essential. (1, 2)

Continuing agitation and persistent inability to fall asleep during withdrawal may also be treated symptomatically by using the antidepressant trazodone (Desyrel). Diphenhydramine (Benadryl) can also be used for its sedating properties. (1, 2)

Common physiological symptoms of chronic stimulant abuse/dependence

- Extreme fatigue—with physical and mental exhaustion and disrupted sleep patterns
- Nutritional disorders—extreme weight loss, anemia, anorexia, cachexia (body wasting)
- Poor hygiene and self-care
- Skin disorders and secondary skin infections—itching, lesions, hives, urticaria
- Hair loss
- Muscle pain/tenderness—may indicate rhabdomyolysis
- Cardiovascular damage—from toxicity and contaminants in MA production, with concomitant renal and hepatic problems
- Hypertensive crises with renal damage from sustained hypertension
- Difficulty breathing—may reflect pulmonary edema, pneumonitis, obstructive airway disease, barotrauma, and other complications
- Myocarditis, infarcts
- Headaches, strokes, seizures, vision loss
- Choreoathetoid (involuntary movement) disorders
- Impaired sexual performance and reproductive functioning
- Cerebrovascular changes, including evidence of cerebral hemorrhages and atrophy with associated cognitive deficits
- Ischemic bowel, gastrointestinal complaints

Common psychological/behavioral symptoms of chronic stimulant abuse/dependence

- Paranoia—with misinterpretation of environmental cues; psychosis with delusions, and hallucinations
- Apprehension—with hopelessness and fear of impending doom that resembles a panic disorder
- Depression—with suicidal thinking and behavior
- Acute anxiety
- Eating disorders

Distinctive indicators of chronic stimulant abuse/dependence

- Nasal perforations and nose bleeds among snorters
- Dental problems, including missing teeth, bleeding and infected gums, dental caries
- Muscle cramping related to dehydration with low magnesium and potassium levels
- Dermatitis around the mouth from smoking hydrochloride salt
- Stale urine smell due to ammonia constituents used in manufacturing MA
- Various dermatologic conditions, including excoriated skin lesions
- Serious constipation due to dehydration and insufficient dietary fiber

Reducing the risk of violence

Medical personnel must be prepared for the paranoia, aggression, and violence that often accompany stimulant use. These personnel should

- Keep the client in touch with reality by identifying themselves, using the client's name, and anticipating his concerns. (2)
- Place the client in a quiet, subdued environment with only moderate stimuli. Ensure sufficient space so that the client does not feel confined. Have the door readily accessible to both the client and the interviewer, but do not let the client get between the interviewer and the door. (2)
- Acknowledge agitation and potential for escalation into violence by reassuring the client that they are aware of his distress; asking clear, simple questions; tolerating repetitive replies; and remaining nonconfrontational. (2)
- Foster confidence by listening carefully, remaining nonjudgmental, and reinforcing any progress made. (2)
- Reduce risk by removing objects from the room that could be used as weapons and discreetly ensuring that the client has no weapons. (2)
- Be prepared to show force if necessary by having a backup plan for help and having chemical and physical restraints immediately available. (2)
- Train all medical or emergency staff to work as a team in managing volatile clients. (2)

There are a number of medical and psychiatric disorders that frequently accompany stimulant abuse and dependence. An awareness of these conditions is important for the safe and effective treatment of stimulant disorders. The conditions include

- Cardiovascular system effects
- Respiratory–pulmonary effects
- Cerebrovascular complications
- Muscular and renal toxicity
- Gastrointestinal complaints
- Infections
- Effects on reproduction/formation of fetus/newborn children
- HIV/AIDS and hepatitis
- Toxic psychosis
- Aggression and violence
- Polysubstance abuse
- Traumatic injury

Assessment and diagnosis

A diagnosis can be based on established DSM-IV criteria for amphetamine or cocaine use/abuse/dependence and other listed composites. (1)

An appropriate substance use history should include the substance(s) and medications used during the last 30 days; the specific substance(s) or combinations typically used with the usual dose, frequency, and route of administration; the duration of use/abuse; and the time and amount of last use as well as when the symptoms or complaints developed and how they have progressed. (2)

Stimulants typically can be detected in urine for approximately 24 to 48 hours following use and, maximally, for 3 days.

Special Groups and Settings

The Consensus Panel feels strongly that cultural competence in treatment extends beyond racial/ethnic sensitivity to understanding the mores of groups bound together by gender, age, geography, sexual preferences, criminal activity, substance use, and medical and mental illnesses. The Consensus Panel therefore recommends the following:

- Counselors should be trained in cultural sensitivity and cultural competency issues to enhance the counselor's understanding and appreciation of both the client's background and his needs within that context. (1, 2)
- Intravenous drug users should have access to multicomponent HIV prevention programs, which include instruction on bleach disinfection along with skills training, counseling, and HIV testing. Needle

exchange programs may also be helpful. (1, 2)

- For counselors working with gay men, education of the sexual and social behaviors that are common among this population (including the widespread use of MA), as well as the stigma associated with substance abuse in the gay community, should be available. (2)
- For clients in narcotic replacement treatment, including methadone and LAAM, cocaine use is a major clinical problem. The most effective method of addressing this particular community appears to be contingency management approaches. (2)
- Clients with co-occurring psychiatric disorders have high levels of stimulant abuse and dependence. Successful treatment of these individuals requires close coordination of psychiatric and stimulant use disorder treatments. (2)
- Treatment for individuals in the criminal justice system is a rapidly expanding area of need. Stimulant users represent a substantial portion of the individuals in the court and prison treatment population. (2)
- For rural populations, forming linkages between social service agencies, providing treatment services that are flexible in scope and structure, and using nontraditional outreach sites such as mobile or satellite offices are all important interventions. (2)
- Counselors should be aware of the special needs of women and adolescents, including domestic issues, medical problems, child care needs, academic performance, and so on. Gender-specific treatment groups and school-based clinics can be helpful in reaching these particular groups. (1, 2)

Conclusion

In stimulant use disorder treatment today, providers have the opportunity to move the role of scientifically based approaches into the forefront of the treatment effort. Recent findings from basic and clinical research serve as the foundation of the evolving treatment system for stimulant use disorders and have yielded an entirely new set of strategies and tools to assist in the treatment of stimulant-related clinical disorders.

As knowledge of stimulants and brain functioning continues to grow, new approaches are likely to be forthcoming.

The development of pharmacotherapies for the treatment of stimulant use disorders is a major priority of current research efforts, and it is likely that these efforts will provide some important new options in the near future. As these new treatments are introduced into the service delivery system and integrated into mainstream care, it will be essential for training tools, including this TIP, to be regularly updated.

1 Introduction

In the early 1980s, thousands of people began to seek treatment to help them with their struggle to stop using cocaine. The U.S. health care system was rapidly overwhelmed. To many treatment experts who had spent their careers treating heroin addicts and alcoholics, the idea that someone would require "treatment" to discontinue cocaine use was a novelty. Among the first questions asked of the individuals seeking treatment were, "What do you need treatment for?" and "Why don't you just stop using?" Today, much more is known about addiction to cocaine and other stimulants. Although researchers, clinicians, and treatment providers have gained insights into why it is so difficult for stimulant users to stop using and why they need treatment, it is only recently that the substance use disorder treatment field has determined the most appropriate treatment approaches for these individuals.

When the U.S. cocaine epidemic was just beginning, there was a generally held assumption, even among addiction experts, that cocaine was not "truly addicting." A popular joke during this period was that "cocaine is God's way of telling you that you have too much money."

The cocaine epidemic that began in the 1970s peaked in the 1980s and slowly declined in the mid 1990s (Golub and Johnson, 1997). The pattern was similar to the first epidemic that occurred 30 years after cocaine hydrochloride was first isolated from coca leaves in 1885. During the first epidemic, physicians mistook cocaine's powerful stimulant properties as a cure for depression, morphine addiction, chronic tuberculosis, and a long list of other disorders. Physicians and other "healers" prescribed the drug for a range of maladies, and cocaine soon became the major active ingredient in many popular medicines, tonics, and elixirs (including the original formulation of Coca-Cola®).

Eventually, however, the adverse effects of high-dose and consistent use were recognized. This recognition soon led to legislative responses. First, the Pure Food and Drug Act of 1906 required the proper labeling of cocaine "and other narcotics" on proprietary medicines. Second, the Harrison Act of 1914 virtually eliminated the use of cocaine-containing patent medicines by forbidding their manufacture and sale. But cocaine did not simply go away, and sometime after 1970, a complex set of social and economic circumstances conspired to prompt its return. Increased demand for the drug initially drove supply, and subsequently, its widespread availability and reduced cost fostered greater demand and abuse.

The cocaine epidemic of the 1980s and early 1990s affected a broad spectrum of American society, with the advent of crack cocaine hitting major cities the hardest. A less publicized and more geographically circumscribed stimulant epidemic is the rise of methamphetamine (MA) in the West and Midwest. The spread of MA has brought many of the health, legal, and social problems like those associated with cocaine to smaller and more rural communities.

These stimulant epidemics have had a devastating impact on American society. The impact of illicit stimulant abuse has affected international politics, the U.S. legal system, and the U.S. health care system. "Freebasing," "crack houses," and "coke fiend" have all entered the American lexicon to describe elements of the stimulant epidemic. As the end of the 20th century nears, the powerful psychostimulants cocaine and MA and their derivatives have joined opiates and alcohol as primary targets in the efforts to combat substance abuse and dependence. But on the positive side, the pressing need to effectively deal with stimulant epidemics and treat people with stimulant use disorders has produced a tremendous amount of scientific and clinical research. The results of this research have broadened our knowledge of the human brain and expanded our understanding of substance use disorders.

The slow response of major U.S. institutions to the dangers of cocaine and MA was partly due to an ignorance of the basic biological and psychological effects of these powerful psychostimulants. The knowledge gained over the past two decades on the properties of these substances can help treatment providers and other health professionals to understand, prevent, and treat the problems created by the use and abuse of cocaine and MA. This Treatment Improvement Protocol (TIP) summarizes the latest research as well as first-hand clinical experience of substance use disorder treatment professionals.

Purpose of the TIP

Since the mid-1980s, there has been an explosion of knowledge about the effects of cocaine and MA. Because these psychostimulants alter the functioning of the body and the brain so profoundly, physicians, nurses, psychologists, social workers, marriage and family counselors, and substance abuse counselors must understand the profound biological aspects of stimulant addiction. New areas of expertise include the relevant pharmacology, neurobiology, psychiatric and psychological manifestations, and appropriate treatment approaches for stimulant abuse and dependence. The new findings suggest that neurological impairments may last up to 2 years after cessation of stimulant use (Hoff et al., 1996; Melega et al., 1997a).

This TIP presents current knowledge of the nature and treatment of stimulant use disorders. The TIP is designed to be a resource that provides scientifically established information and presents it in a manner that makes it available and relevant for both clinicians and "front line" treatment providers. In addition, the document reviews what is known about treating the medical, psychiatric, and substance abuse/dependence problems associated with the use of cocaine and MA. The treatment section emphasizes those approaches that have been established with empirical support. However, because the field of treating stimulant use disorders is barely a decade old, a set of treatment techniques supported by leading addiction specialists has been included after review and synthesis by the members of the Consensus Panel.

Importance of Science in Building the Treatments Of the Future

The Consensus Panel believes that scientifically derived knowledge should serve as the foundation of treatment for stimulant use disorders. Findings from basic and clinical research efforts funded by the National Institute on Drug Abuse (NIDA), as well as other government and private institutions, have given treatment providers an entirely new set of strategies and tools to assist those with stimulant-related clinical disorders. The field of stimulant use disorder treatment presents the perfect opportunity to move the role of scientifically based approaches into the forefront of the treatment effort. There is very little in the way of a "traditional treatment system" for stimulant use disorders, and therefore, there should be fewer "turf battles" over the implementation of new treatment approaches.

The Consensus Panel recognizes that most traditional treatment approaches are still viable and highly regarded by providers, and that new treatment techniques may be initially viewed with distrust. Continuing research and clinical experience will ultimately reveal the efficacy of such treatments.

At this time, the approaches with the greatest empirical support are a variety of psychosocial–behavioral strategies, delivered in outpatient settings. However, as knowledge of stimulants and brain functioning rapidly increases, thanks to active research funded by Federal agencies and private foundations, new approaches will soon be forthcoming. The development of pharmacotherapies for the treatment of stimulant use disorders is a major priority of the current research efforts, and it is likely that these efforts will provide some important new options in the near future.

Scope of the TIP

For purposes of this TIP, the substances included in the category of "stimulants" include the derivatives of the coca plant (cocaine hydrochloride and its derivatives) and the synthetically produced amphetamines, with emphasis on the major illicitly produced and abused drug of this category, MA (in its various forms). Certainly there are other stimulants that are more widely used (e.g., caffeine) and that produce major health and social problems (e.g., nicotine); however, an extensive discussion of issues associated with these substances is beyond the scope of this document. Although considered drugs of abuse, MA analogs—compounds with similar molecular structures but not necessarily similar effects, sometimes called "designer drugs"—such as MDA (3,4-methylenedioxy-amphetamine) and MDMA (3,4-methylene-dioxymethamphetamine)—have not been studied adequately for inclusion in this document.

A Brief History of Stimulant Use in the United States

Cocaine

Cocaine hydrochloride is extracted from the leaves of the coca plant (*Erythroxylon coca*), which is indigenous to the Andean highlands of South America. In its extracted and purified form, it is one of the most potent stimulants of natural origin (Drug Enforcement Agency [DEA], 1995). For thousands of years, the Native Americans in the Andean region have chewed coca leaves to relieve fatigue, much as present-day Americans chew tobacco. Just as tea and coffee are brewed as refreshments or "pick-me-ups," the Andean natives brewed coca leaves into a tea. Furthermore, Andean groups have historically burned or smoked various parts of the coca plant as part of their religious and medicinal practices (Siegel, 1982). However, none of these other uses has had the same impact as purified cocaine hydrochloride.

German chemist Albert Niemann recognized the stimulant properties of the cocaine plant, and in the mid-1800s (ca. 1862) extracted the pure chemical, cocaine hydrochloride. In the early 1880s, the drug's anesthetic properties were discovered, and it was soon used in eye, nose, and throat surgery. As physicians and other prescribers became aware of cocaine's psychoactive properties, it was widely dispensed for anxiety, depression, and addiction treatment (primarily for morphine use).

Extravagant claims of its curative powers increased cocaine's popularity; by the early 1900s, it was the main active ingredient in a wide range of patent medicines, tonics, elixirs, and fluid extracts. It is believed that the original formula of Coca-Cola® that was developed in 1886 by Georgia pharmacist John Pemberton contained approximately 2.5 mg of cocaine per 100 mL of fluid (*Coca-Cola Bottling of Shreveport, Inc., et al., vs. The Coca-Cola Company, a Delaware Corporation*, 769 F.Supp.671). This formula was sold as a headache cure and stimulant. Another pharmacist bought the rights and founded the Coca-Cola Company in 1892.

By the early 1900s, public health officials were becoming alarmed by the medical, psychiatric, and social problems associated with excessive cocaine use. These concerns from health officials and legal authorities played a major role in initiating and supporting the effort to pass the Harrison Narcotic Act of 1914. This Federal legislation severely restricted the legal uses for cocaine and, for all practical purposes, ended the extensive use and abuse of cocaine in the early part of the 20th century. Interestingly, cocaine hit a low during the 1930s when the advent of amphetamine almost eradicated demand.

From the time of the Harrison Narcotic Act until the 1970s, cocaine use was generally limited to groups on the periphery of society. Legal prohibitions and severely restricted supplies of the drug helped to maintain its low profile. But microcultures of cocaine snorters, swallowers, and shooters remained, and cultivation of coca plants continued in the South American countries that traditionally grew them—Bolivia, Peru, Colombia, and Ecuador.

As the cultural proscriptions against the use of drugs for recreational purposes weakened during the 1960s, cocaine again became part of the American drug scene. Its use increased along with the use of many other psychoactive substances. Snorting was the initial mode, and most experimenters were occasional consumers. They experienced the cocaine euphoria and generally went back to their "normal" lives. Because of this casual use, the fictitious notion arose that cocaine was harmless.

In the 1960s, limited supplies and high prices combined to restrict the use of cocaine to relatively small amounts used by a small number of individuals. Although serious clinical problems were being connected with the use of hallucinogens, barbiturates, and amphetamines, little attention was given to the problems associated with cocaine use because they were rarely seen.

As recently as the late 1970s, many experts and public health officials believed that cocaine was a relatively benign substance and primarily of interest as a "recreational" drug. It was thought that only those who had access to very large supplies of the drug and/or those who were somewhat mentally unstable were at risk for developing problems with cocaine. A notable exception among these experts was the voice of two San Francisco addiction experts who sounded a prophetic warning about cocaine:

> In summary, cocaine is a central nervous system stimulant of moderately high abuse potential. At the present time the preferred route of administration is intranasal and the dosage patterns are relatively low. The social rituals surrounding the drug endorse primarily recreational use while the high cost and low availability of the drug produce the current low rate of cocaine abuse in the United States....Most users now use cocaine by the intranasal route at moderately low dosages, while a relatively small percentage use cocaine intranasally or intravenously at high dosages. However, if the drug were more readily available at a substantially lower cost, or if certain socio-cultural rituals endorsed and supported the higher dose patterns, more destructive patterns of abuse could develop. (Wesson and Smith, 1977, pp. 149–150)

Within 5 years of the observation by Wesson and Smith, both essential developments they predicted had occurred. The production of coca in South America expanded from a cottage industry of small groups of subsistence farmers into a major agricultural business that was financed by organized families or "cartels." The manufacture and trafficking of cocaine became a multibillion dollar industry, with profit margins high enough that governments and entire legal systems became corrupted by the influx of cocaine industry money. Supplies of cocaine into the United States increased exponentially. During the early to mid-1980s, according to DEA reports, the estimated amounts of cocaine entering the United States doubled and tripled year after year. These supplies of cocaine made the drug available in purer form and at a more affordable cost to consumers.

Cocaine hydrochloride is generally distributed as a white crystalline powder or as an off-white chunky material. The powder form is usually snorted intranasally. As cocaine became plentiful and less expensive in the early 1980s, its users began to experiment with its various forms and with different routes of administration. Some users began to smoke the powder form by mixing it with tobacco or marijuana. However, those who smoked the powder reported little if any intoxication.

At the same time, users in South America began to smoke *base* (coca paste), which is one of the products from which cocaine powder is derived (Siegel, 1987). Coca paste is more concentrated than the powder form. Paste smokers report immediate intoxication, with effects similar to those reported by intravenous users. The first hospital admissions for adverse effects of coca paste smoking were in Peru in 1972 (Jeri, 1984). The practice of smoking coca paste appears to have traveled to other countries via illicit cocaine trafficking corridors.

Drug traffickers in the United States learned of the effects of smoking base, but they confused its preparation with that of cocaine freebase, in which the cocaine alkaloid in cocaine hydrochloride is "freed" from the other components (Siegel, 1982). So it was quite by accident that this new process of "freebase" cocaine was discovered. However, its properties were quite unlike those of either coca paste or cocaine powder. Freebase cocaine does not dissolve easily in the blood or mucous membranes of the nasal passages, but it is readily volatilized and can be effectively smoked. The phenomenon of smoking this freebase form was first reported in California in 1974, and by 1980, its use was reported throughout the United States (Siegel, 1982). Today, chunks of the freebase form are most often known as "rock" or "crack."

The next phase in the American cocaine epidemic came when cocaine traffickers saw an opportunity to expand the retail market by delivering to the consumer smaller, more affordable packages of the drug. Chunks of rock cocaine were soon being sold in small glass vials or plastic containers at a cost of $10 to $20. This new retailing effort made a product that was extremely desirable and inexpensive readily available to a much wider user base. The strategy worked extraordinarily well for the cocaine industry.

By late 1985 and early 1986, the retailing of freebase cocaine had swept through most urban centers of the United States. This form was introduced into new markets by highly organized and sophisticated distribution networks. In an effort to make the product distinctive, it was marketed under the new name "crack." There are numerous versions of the origin of the term "crack," but the most likely is that as the freebase cocaine is being heated and volatilized into its smokable form, it makes a characteristic crackling or popping sound.

The crack epidemic was at its worst from 1985 through the end of the decade, although it still remains a serious health and social problem. The introduction of crack into urban communities produced devastating consequences. Health-related problems, rapidly escalating rates of addiction, and an extraordinary wave of street crime and property crime swept through most major American cities. In many areas, street gangs of young males were central to the distribution and sales of crack. Warfare between street gangs battling over turf resulted in many fatalities among gang members as well as innocent bystanders in the community. As drug-related crime escalated dramatically, legal penalties for sales of cocaine and crack were increased, and U.S. jails and prisons rapidly filled with crack users, dealers, distributors, and those involved in the violence associated with the crack trade.

At the peak of the cocaine epidemic, a conservative estimate in the mid-1980s suggested that as many as 8 million Americans used cocaine regularly and that 5 to 8 percent of them had developed a serious cocaine dependence (Cregler and Mark, 1986). The 1988 National Household Survey on Drug Abuse (NHSDA) found that the number of heavy crack and cocaine users rose significantly from 1985 to 1988 (Substance Abuse and Mental Health Services Administration [SAMHSA], 1988). During this period, there was a 33 percent increase among those using crack or cocaine once a week or more; for those using crack or cocaine on a daily or near-daily basis, the rate rose by 19 percent (SAMHSA, 1989).

By the mid 1980s, the use of crack cocaine had replaced heroin use as the main illicit drug problem in the United States. According to the 1997 NHSDA, the number of Americans who used cocaine within the preceding month of the survey numbered about 1.5 million; occasional users (those who used cocaine less often than monthly) numbered approximately 2.6 million, down from 7.1 million in 1985 (SAMHSA, 1998). Only recently have researchers been able to demonstrate a clear decline or stabilization in the use of crack cocaine in U.S. cities (Golub and Johnson, 1997).

Methamphetamine

Amphetamine, the predecessor to MA, was first synthesized in 1887 and became commercially available in 1932 as a nasal spray for the treatment of asthma (Beebe and Walley, 1995). Amphetamine's stimulant properties were soon recognized, which led to additional medical and functional applications. By 1937, amphetamine was available by prescription to treat the sleep disorder narcolepsy and the syndrome that is now called attention deficit/hyperactivity disorder (AD/HD). After the introduction of amphetamine, other more potent forms were developed and made readily available to the public. These new forms included dextroamphetamine sulfate (Dexedrine) and methamphetamine (Methedrine). Because of their stimulant properties, these new forms were also used to enhance performance. During World War II, MA was widely used by soldiers to fight fatigue and enhance performance. Pilots used MA to stay awake for long periods of time. After World War II, intravenous MA abuse reached epidemic proportions in Japan, when supplies stored for military use became available to the public.

During the 1950s, truckers often used legally manufactured tablets of MA to stay awake on long hauls, athletes used them to enhance performance, and students used them to study long hours and maintain busy schedules. The use of these stimulants during this period was typically not associated with the concept of substance abuse. Although the drugs required a medical prescription, their nonmedical use was generally considered to be simply a method of enhancing performance and usually did not lead to severe addiction. This pattern changed drastically in the 1960s with the increased availability of injectable MA. Intravenous abuse spread among a subculture known as "speed freaks." Evidence soon began to mount that the dangers from the abuse of MA and amphetamine outweighed most of their therapeutic uses.

Eventually, many pharmaceutical amphetamine products were removed from the market, and doctors prescribed the remaining products less freely. As the supply of amphetamine and MA decreased, demand in the black market soon increased, which led to increased illicit production of the drugs. In 1965, greater control measures to curb the trafficking in amphetamine began with amendments to Federal food and drug laws, and the 1970 Controlled Substances Act severely restricted the legal production of these stimulants. Throughout the 1970s, the production and distribution of MA declined nationwide. It was, however, still concentrated in a few cities or regions.

For a number of reasons, the 1970 Controlled Substances Act did not succeed in eliminating MA use. First, the materials and equipment required to produce MA are inexpensive. Second, MA is relatively easy to manufacture. Finally, the active ingredients needed to prepare the drug are relatively easy to obtain. Moreover, clandestine manufacturers developed alternative methods of MA production that were not covered under the law. Furthermore, compared with other stimulants such as cocaine, MA is cheaper and its effects longer lasting.

As the demand for MA increased, so did its production in clandestine laboratories. Because two of the precursor drugs (ephedrine and pseudoephedrine) used to manufacture MA are widely available in Mexico and are easily smuggled into the United States, the clandestine manufacture of MA was initially based in the West and Southwest. In this region, the production and distribution of MA have been historically associated with motorcycle gangs (Feucht and Kyle, 1996).

By the mid-1980s, the number of illegal, makeshift MA laboratories in rural communities in western States had mushroomed, especially in California. In certain areas of California (e.g., San Bernardino, San Diego, San Francisco, and Riverside counties), the problem with MA far outdistanced problems with cocaine even as early as the mid-1980s (Huber et al., 1997). These areas were especially saturated with MA because the illicit laboratories set up to manufacture MA were located in the rural desert areas in proximity to these Southern California urban and suburban areas. The manufacture of MA in these rural regions was preferred by drug manufacturers because the "cooking" process of MA produces a very strong chemical smell, making the home laboratories easy to detect. This phenomenon was evidenced by the tremendous number of MA laboratories seized by law enforcement officials in these locations in the latter half of the 1980s.

There were other indicators of MA's continued spread. For example, data from the NHSDA (SAMHSA, 1988, 1989) revealed that 1 in 10 Californians had used MA at least once in their lives, and that 1 in 50 had used MA within the 12 months prior to the survey. Along with the general increases in MA use, striking increases were observed in MA-related hospital admissions, seizure activity, and deaths (Anglin et al., 1998).

In a further attempt to curb MA production, the Chemical Trafficking and Diversion Act of 1988 amended the 1970 legislation to require wholesalers to record imports and exports of some of MA's chemical precursors, including ephedrine, pseudoephedrine, phenylacetic acid, benzyl cyanide, and benzyl chloride. However, these chemicals could still be easily obtained outside the United States. In particular, the continued availability of precursor chemicals in Mexico resulted in increases in illicit production there, and increasing amounts of MA were smuggled into the United States. Today, Mexican drug cartels have increased their share of the U.S. wholesale MA market (Feucht and Kyle, 1996).

By the late 1980s, MA had spread to other areas of the United States. In Hawaii, MA was being smuggled in from Taiwan and South Korea, and by the summer of 1988, MA was relatively widespread throughout that State. By 1990, MA was being distributed from Hawaii to the U.S. mainland. According to information available from the Drug Abuse Warning Network (DAWN), the estimated number of nationwide emergency room substance abuse episodes involving MA has increased steadily since 1992 (SAMHSA, 1996b). In fact, the number of MA-related episodes recorded during 1994 was almost double the number of episodes in 1989. DAWN statistics indicate the areas hardest hit by MA abuse include San Diego, Phoenix, Los Angeles, Dallas, Denver, and Seattle (see Figure 1-1).

MA—which is known by many street names (see Figure 1-2)—can be swallowed in pill or tablet form, snorted (intranasally) in its powdered form, or injected intravenously in its solution form. Of these three routes of administration, injection leads to the quickest and most intense effects, what users call "the rush." However, MA can be transformed into a high-purity solid form that appears as clear, large, chunky crystals known as "ice" or "glass." This form of MA can be smoked, and because it is a more powerful form, the rush can be immediate and more intense than when administered intravenously. The euphoria reportedly lasts longer than that of smoked crack cocaine.

The ice form of MA first appeared in Hawaii and soon thereafter on the West Coast in the late 1980s. It has been reported that the method of manufacturing and processing MA into ice was imported from the Philippines. The extensive use of MA in some parts of Oahu, especially Honolulu, produced extremely serious concerns about the violence and crime associated with ice use. Ice has continued to be a preferred form of the drug in Hawaii for over a decade. Although health officials on the mainland worried that ice would become a major problem, its availability currently appears to be limited to Hawaii and to some Asian-American communities in Seattle and California (Office of National Drug Control Policy [ONDCP], 1998b). At the time of this writing, it is believed that ice trafficking and availability are very limited in the rest of the United States.

Figure 1-1

MA-Related Deaths for Los Angeles, Phoenix, San Diego, and San Francisco

City	1992	1993	1994	Percentage Change 1992 to 1994
Los Angeles	68	198	219	+222
Phoenix	20	63	122	+510
San Diego	97	110	172	+77
San Francisco	48	62	69	+44

Source: DEA, 1996.

Figure 1-2
Some Street Names for Methamphetamine

- Crank
- Crypto
- Crystal
- Crystal meth
- Meth
- Quill
- Speed
- Tweak (an MA-like substance)
- White cross
- Yellow bam

For Smokable Forms

- Cristy
- Hanyak
- Ice
- L.A. glass
- Quartz

Source: ONDCP, 1995.

The Current Situation

Cocaine

The common perception today is that the cocaine epidemic of the 1980s and early 1990s is in decline and that the "war on cocaine" has been won. The most recent data from the Drug Use Forecasting (DUF) Program (National Institute of Justice [NIJ], 1997b) confirm that in 12 of the 23 DUF program cities, the crack epidemic was in decline by 1996. A substantial decline of at least 10 percent in the overall rate of detected crack/cocaine use was observed in Cleveland, Dallas, Detroit, Houston, Los Angeles, New Orleans, Philadelphia, San Diego, San Jose, and Washington, D.C. Other locations exhibited substantial declines in detected crack/cocaine use among youths, which suggested declines in the overall rate were forthcoming.

Other data, however, indicate that the notion of a declining cocaine epidemic is a misperception. According to the 1996 DUF data (NIJ, 1997b), the crack epidemic in some cities rages on as strong as ever. These locations include Atlanta, Denver, Indianapolis, Phoenix, and St. Louis. There were also some remarkable increases in cocaine-positive tests among male arrestees. In Omaha, the rate of positive tests was 24 percent in 1996, up from 19 percent in 1995. In Miami, cocaine positives increased from 42 to 52 percent. In Indianapolis, cocaine-positive tests grew by 3 percent.

Other studies also indicate that cocaine use is still at high levels. In 1997, NHSDA estimated that approximately 1.5 million Americans used cocaine in the past year (SAMHSA, 1998). Of the 18- to 25- year olds surveyed, 8.9 percent reported using cocaine, and of those age 26 to 34, 18.4 percent reported using cocaine.

A recent annual high school survey, the 1997 Monitoring the Future Study, reports that the use of cocaine, in any form, continues to climb (NIDA, 1998b), whereas cocaine remains the most prevalent drug in the DUF monitoring system (NIJ, 1997b). Figure 1-3 shows the percentage of high school seniors who reported using cocaine and other stimulants.

In 1996, the Drug Abuse Warning Network (SAMHSA, 1996b) reported 487,600 drug-related episodes in hospital emergency departments nationwide. Of these, approximately 20 percent were related to the use of cocaine or crack.

According to *Pulse Check* (ONDCP, 1997b), the market for cocaine/crack appears to have stabilized, but in many areas, the drug remains in great demand. There have been reports of rising cocaine use in specific communities: the suburbs of Birmingham, Alabama, the Hispanic-American community along the Texas border, and young people in the New York/New Jersey area. In several areas, there is the reported reemergence of powder cocaine.

Methamphetamine

Unlike the cocaine/crack epidemic of the 1980s and 1990s, the MA epidemic has been regional. But in those areas, MA has taken a strong hold. For example, in some areas of the country, MA has surpassed both alcohol and cocaine as the primary substance of abuse among treatment admissions (Center for Substance Abuse Research, 1997; CSAT, 1997). According to the DEA, MA trafficking and use in the United States have been on the rise over the past few years, as indicated by investigative, seizure, price, purity, and abuse data (DEA, 1996). Recent reports of increased MA use are mentioned in several monitoring and forecasting studies (e.g., DAWN, Monitoring the Future, NHSDA, DUF, and the Treatment Episodes Data Set System).

Increases in the seizures of MA serve as an additional measure of the increasing problem (ONDCP, 1996; CSAT, 1997). For example, in 1995, both the number and weight of MA seizures were the highest in over a decade (CSAT, 1997). In Community Epidemiology Work Group (CEWG) cities, MA-related deaths have been steadily increasing (CEWG, 1996a, 1996b). From 1991 through 1994, MA-related emergency department (ED) episodes increased 256 percent (SAMHSA, 1996a), involving more than 17,000 individuals. DAWN data revealed that MA-related ED episodes had increased by 75 percent from 1993 to 1994.

Although recent reports still place the bulk of MA use in western States including Hawaii (NIJ, 1997b; ONDCP, 1997a), concern continues to grow that the increased number of seizures of clandestine operations and MA-related fatalities in other parts of the country may signal an impending MA epidemic. MA trafficking is increasing in the Southwest, the Midwest, and some southeast regions of the United States (DEA, 1996). In 1996, the DEA seized more clandestine MA manufacturing laboratories in Missouri than in any other State (Samber, 1997). It is reported that MA abuse is very prevalent in several U.S.-affiliated territories in the Pacific (e.g., Guam, the Northern Marianas). According to *Pulse Check* information, MA is increasing in popularity in Atlanta and Seattle, and it is listed as an "emerging drug" for Austin, Texas; Washington, D.C.; and Columbia, Maryland (ONDCP, 1997b).

There is also a growing amount of anecdotal information on MA's spread from the West Coast. Recent information from the 1996 National Narcotics Intelligence Consumers Committee Report, which describes law enforcement data on drug-related arrests and seizures, indicates that in the last 2 years, MA has increased significantly in the Southeast and the Midwest (DEA, 1997). Although there is little current empirical data to indicate the spread of MA into more rural heartland areas, there are indicators and anecdotal evidence that

Figure 1-3

1997 Monitoring the Future Study: Drug Use Among High School Seniors

Drug	Percentage ever used	Percentage used in the past year	Percentage used in the past month
Cocaine	8.7	5.5	2.3
Stimulants	16.5	10.2	4.8

Source: NIDA, 1998b.

the manufacture and abuse of MA are having a costly effect in States such as Montana and Iowa (e.g., Kirn, 1998). In fact, the extent of the MA epidemic may be far greater than many experts currently recognize.

Former CSAT Acting Director Camille T. Barry has stated that the particular problem groups for MA abuse are women, gay men, and Asian-Pacific Islanders (Barry, 1998). On a national level, approximately 80 percent of those treated are white Americans. In areas such as Arizona and Minnesota, there has been increasing use in Hispanic and Native American populations. In West Coast cities, the use of MA is increasing among gay men, and use by that population is closely associated with sexual practices (Shoptaw et al., 1997).

Concerns that levels and effects of MA abuse will replicate those of the crack epidemic have put this drug under intense national scrutiny. In response to this concern, The White House launched an initiative called The President's National Strategy for Combating Methamphetamine Abuse. This comprehensive national strategy involves enhanced law enforcement efforts, regulation of precursor chemicals, international initiatives, tougher criminal penalties, legislative proposals, and training of investigators and prosecutors, as well as treatment, prevention, and a public education campaign (ONDCP, 1996). This White House initiative led to passage of the Comprehensive Methamphetamine Control Act of 1996, which in turn created the Methamphetamine Interagency Task Force. The Act set out the Task Force's responsibilities as "designing, implementing, and evaluating the education, prevention, and treatment practices and strategies of the Federal Government with respect to methamphetamine and other synthetic stimulants" (Public Law: 104–237 [10/03/96]).

Several national conferences on MA have been held in the last several years (e.g., CSAT, 1997; ONDCP, 1998c). At a 1997 conference, the ONDCP noted the environmental problems that could stem from MA manufacture:

> Methamphetamine is a synthetic stimulant that can produce extreme aggressiveness and violence. Historically, concentrated abuse of this drug was in the West and Southwest but is now reported to be spreading to the Midwest and the eastern portion of the United States. Methamphetamine production entails extreme environmental risks. Clandestine laboratories produce large amounts of toxic waste, much of which is dumped into the ground or in waterways. The cost to clean up these chemical toxins can easily run into thousands of dollars. (ONDCP, 1998c, p. v)

According to various Federal sources, MA is the most widely used and abused of the amphetamines, including amphetamine and dextroamphetamine sulfate (CSAT, 1997). According to the DEA, MA has been the most prevalent, clandestinely produced controlled substance in the United States since 1977 (DEA, 1996). Because of the recent resurgence in its use, the development of new data on its effects, and its ever-increasing national attention, MA is the only amphetamine reviewed in this TIP.

Summary

During the 1980s and 1990s, the medical, legal, and societal problems created by the importation, manufacture, sale, and use of the powerful psychostimulants cocaine and MA had a tremendous impact on American society. From the devastating crack epidemic in major U.S. urban centers to the MA-produced destruction in small rural communities in the western and midwestern regions of the United States, the damage caused by the stimulant epidemic has been profound. New knowledge about how these substances influence the basic electrical and chemical activity of the human brain has allowed a better understanding of how and why stimulants affect human behavior, and this knowledge has been rapidly absorbed into the development of new treatment efforts. This TIP provides an overview of (1) the new knowledge about stimulants; (2) the treatment efforts to address stimulant abuse disorders; and (3) the other clinical, medical, and social interventions developed in response to the abuse of and dependence on these substances.

2 How Stimulants Affect the Brain and Behavior

Over the last several decades, research on substances of abuse has vastly improved our understanding of human behavior and physiology and the nature of substance abuse and dependence. Basic neurobiological research has enhanced our understanding of the biological and genetic causes of addiction. These discoveries have helped establish addiction as a biological brain disease that is chronic and relapsing in nature (Leshner, 1997). By mapping the neural pathways of pleasure and pain through the human brain, investigators are beginning to understand how abused psychoactive substances, including stimulants, interact with various cells and chemicals in the brain.

This new information has also improved our understanding of appropriate treatment approaches for different substance use disorders. This chapter describes the effects that cocaine and methamphetamine (MA) use have on the user's brain and behavior, which in turn leads to the stimulant users' unique needs. Knowledge of these effects provides the foundation for stimulant-specific treatment approaches. This knowledge will give treatment providers greater insight into stimulant users and why certain treatment approaches are more effective.

Stimulant Abuse And the Brain

According to National Institute on Drug Abuse Director Alan I. Leshner, Ph.D., the fundamental problem in dealing with any substance of abuse is to understand "the target" (i.e., the user). Therefore, to understand why people take drugs such as cocaine and MA and why some people become addicted, we must first understand what these drugs are doing to their target; that is, how stimulants affect the user.

Discussions of substance abuse and dependence often involve some discussion of the root causes—the societal and risk factors that lead to these conditions. To date, investigators have identified as many as 72 risk factors for substance use and dependence (Leshner, 1998). Among them are poverty, racism, social dysfunction, weak families, poor education, poor upbringing, and substance-abusing peer groups. These risk factors—as well as other environmental and genetic factors—only influence an individual's initial decision to use substances of abuse. But after initial use, an individual continues to use a substance because she likes its effects: Use modifies mood, perception, and emotional state. All of these effects are modulated through the brain; in order to understand this phenomenon, it is important to understand some basic neuroscience.

For substances of abuse to exert their effects, they must first get to the brain. The four most common routes of administering psychoactive (mood-changing) substances are (1) oral consumption (i.e., swallowing), (2) intranasal consumption (i.e., snorting), (3) inhalation into the lungs (generally by smoking), and (4) intravenously via hypodermic syringe.

A swallowed substance goes to the stomach and on to the intestinal tract. Some substances easily pass through the digestive tract into the bloodstream. Other substances are broken down into their chemical components (i.e., metabolized) in the digestive system, thereby destroying the substance.

Substances that are inhaled into the lungs adhere to the lining of the nasal passages (the nasal mucosa) through which they enter directly into the bloodstream. Inhaled substances are usually first changed into a gaseous form by igniting (e.g., marijuana) or volatilizing by intense heat (e.g., crack cocaine, the ice form of MA). The lungs offer a large surface area through which the gaseous form may quickly pass directly into the bloodstream.

Injected substances obviously enter the bloodstream directly, although at a somewhat regulated rate. In these last three routes of administration, substances enter the bloodstream in their unmetabolized form.

Once a substance enters the bloodstream, it is transported throughout the body to various organs and organ systems, including the brain. Substances that enter the liver may be metabolized there. Substances that enter the kidney may be excreted. If a female substance user is pregnant, and the substance is able to cross the placenta, then the substance will enter the fetus' bloodstream. Nursing babies may ingest some substances from breast milk.

To enter the brain, a substance's molecules must first get through its chemical protection system, which consists mainly of the blood–brain barrier. Tight cell-wall junctions and a layer of cells around the blood vessels keep large or electrically charged molecules from entering the brain. However, small neutral molecules like those of cocaine and MA easily pass through the blood–brain barrier and enter the brain. Once inside the brain, substances of abuse begin to exert their psychoactive effects.

Fundamentals of the Nervous System

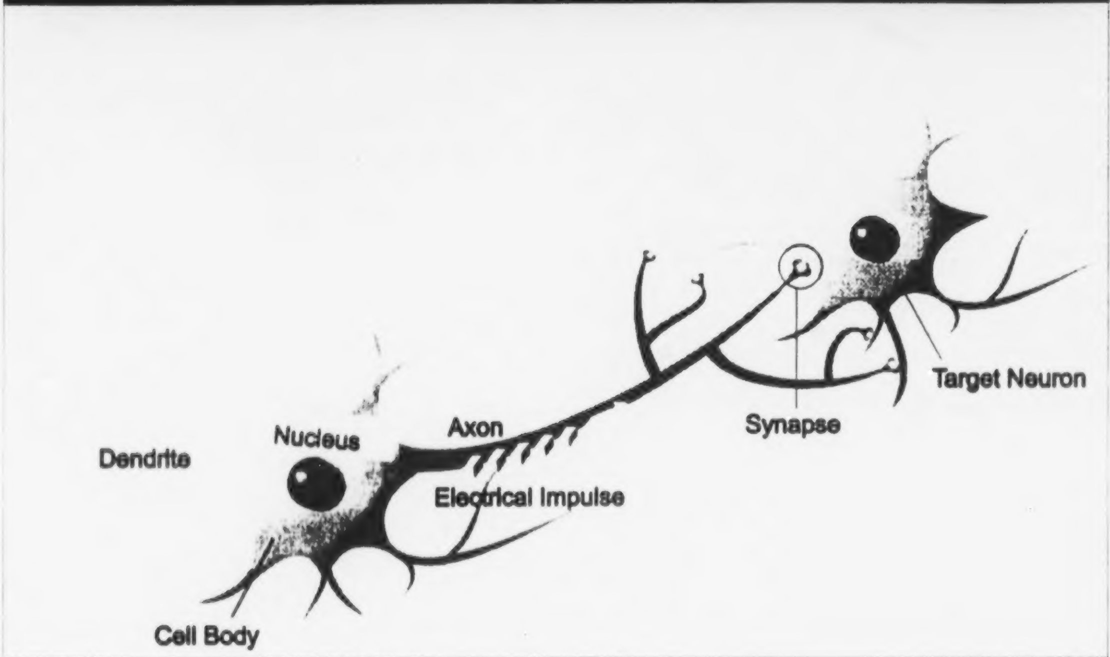
The human nervous system is an elaborately wired communication system, and the brain is the control center. The brain processes sensory information from throughout the body, guides muscle movement and locomotion, regulates a multitude of bodily functions, forms thoughts and feelings, modulates perception and moods, and essentially controls all behavior.

The brain is organized into lobes, which are responsible for specialized functions like cognitive and sensory processes and motor coordination. These lobes are made up of far more complex units called circuits, which involve direct connections among the billions of specialized cells that the various substances of abuse may affect.

The fundamental functional unit of the brain's circuits is a specialized cell called a *neuron*, which conveys information both electrically and chemically. The function of the neuron is to transmit information: It receives signals from other neurons, integrates and interprets these signals, and in turn, transmits signals on to other, adjacent neurons (Charness, 1990).

A typical neuron (see Figure 2-1) consists of a main cell body (which contains the nucleus and all of the cell's genetic information), a large number of offshoots called *dendrites* (typically 10,000 or more per neuron), and one long fiber

Figure 2-1
The Typical Neuron



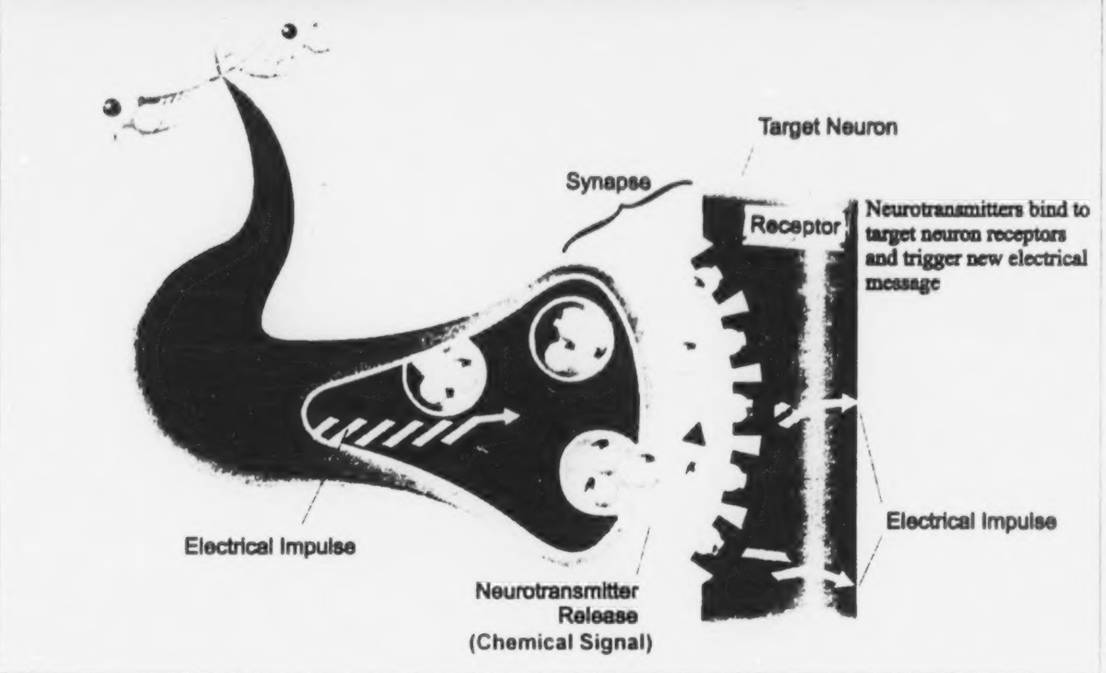
known as the *axon*. At the end of the axon are additional offshoots that form the connections with other neurons. Within neurons, the signals are carried in the form of electrical impulses. But when signals are sent from one neuron to another, they must cross the gap at the point of connection between the two communicating neurons. This gap is called a *synapse*. At the synapse, the electrical signal within the neuron is converted to a chemical signal and sent across the synapse to the target (i.e., receiving) neuron. The chemical signal is conveyed via messenger molecules called *neurotransmitters* that attach to special structures called *receptors* on the outer surface of the target neuron (Charness, 1990). The attachment of the neurotransmitters to the receptors consequently triggers an electrical signal within the target neuron. Approximately 50 to 100 different neurotransmitters have been identified in the human body (Snyder, 1986). Figure 2-2 illustrates a typical synaptic connection and depicts the chemical communication mechanism. Neurotransmitters may have different effects depending on what receptor they activate. Some increase a receiving neuron's responsiveness to an incoming signal—an excitatory effect—whereas others may diminish the responsiveness—an inhibitory effect. The responsiveness of individual neurons affects the functioning of the brain's circuits, as well as how the brain functions as a whole (how it integrates, interprets, and responds to information), which in turn affects the function of the body and the behavior of the individual. The accurate functioning of all neurotransmitter systems is essential for normal brain activities (National Institute on Alcohol Abuse and Alcoholism [NIAAA], 1994; Hiller-Sturmhöfel, 1995).

The Limbic Reward System

The brain circuit that is considered essential to the neurological reinforcement system is called the *limbic reward system* (also called the *dopamine reward system* or the *brain reward system*). This neural circuit spans between the *ventral*

41

Figure 2-2
Typical Synaptic Junction


Target Neuron
Synapse
Receptor
Neurotransmitters bind to target neuron receptors and trigger new electrical message
Electrical Impulse
Electrical Impulse
Neurotransmitter Release (Chemical Signal)

tegmental area (VTA) and the *nucleus accumbens* (see Figure 2-3). Every substance of abuse—alcohol, cocaine, MA, heroin, marijuana, nicotine—has some effect on the limbic reward system. Substances of abuse also affect the nucleus accumbens by increasing the release of the neurotransmitter dopamine, which helps to regulate the feelings of pleasure (euphoria and satisfaction). Dopamine also plays an important role in the control of movement, cognition, motivation, and reward (Wise, 1982; Robbins et al., 1989; Di Chiara, 1995). High levels of free dopamine in the brain generally enhance mood and increase body movement (i.e., motor activity), but too much dopamine may produce nervousness, irritability, aggressiveness, and paranoia that approximates schizophrenia, as well as the hallucinations and bizarre thoughts of schizophrenia. Too little dopamine in certain areas of the brain results in the tremors and paralysis of Parkinson's disease.

Natural activities such as eating, drinking, and sex activate the nucleus accumbens, inducing considerable communication among this structure's neurons. This internal communication leads to the release of dopamine. The released dopamine produces immediate, but ephemeral, feelings of pleasure and elation. As dopamine levels subside, so do the feelings of pleasure. But if the activity is repeated, then dopamine is again released, and more feelings of pleasure and euphoria are produced. The release of dopamine and the resulting pleasurable feelings positively reinforce such activities in both humans and animals and motivate the repetition of these activities.

Dopamine is believed to play an important role in the reinforcement of and motivation for repetitive actions (Di Chiara, 1997; Wise, 1982), and there is an increasing amount of scientific evidence suggesting that the limbic reward system and levels of free dopamine provide the

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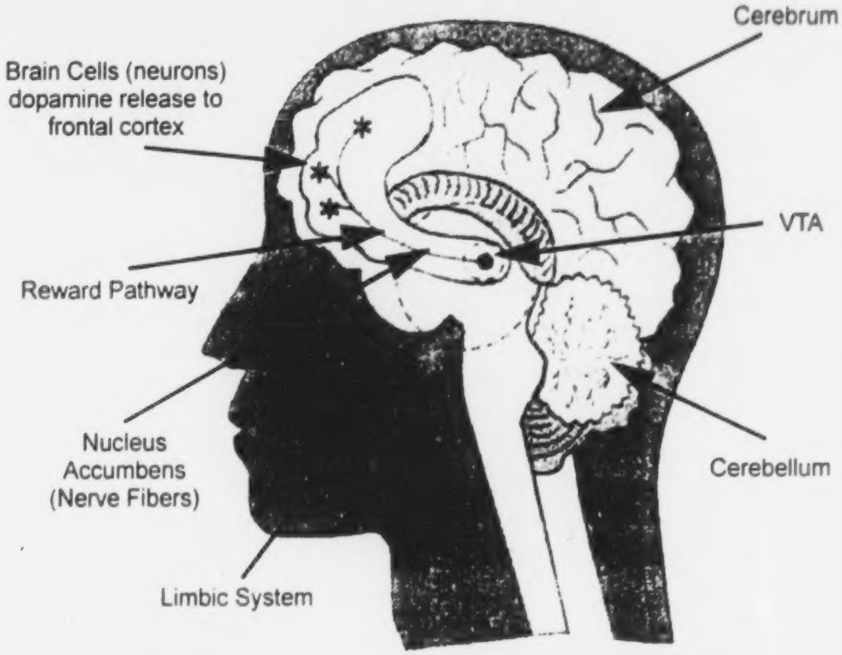
Figure 2-3
The Limbic Reward System


Cerebrum
Brain Cells (neurons) dopamine release to frontal cortex
VTA
Reward Pathway
Nucleus Accumbens (Nerve Fibers)
Cerebellum
Limbic System

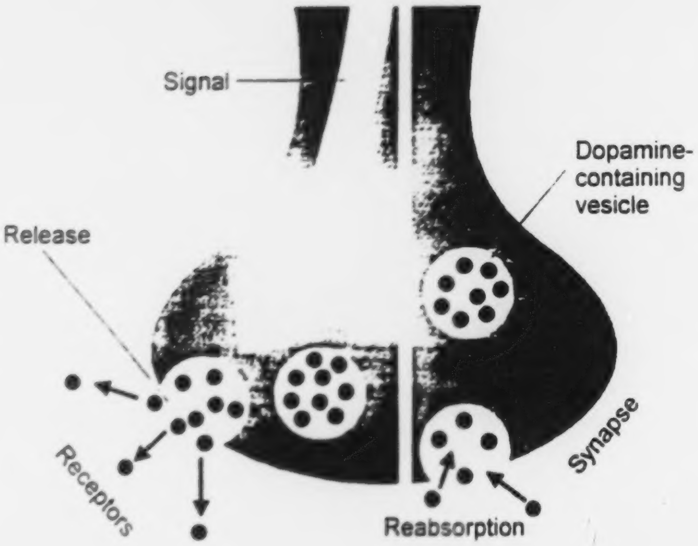
Note: The structures in the limbic reward system that are affected by cocaine and methamphetamine include dopamine neurons in the ventral tegmental area (VTA), which is connected to the nucleus accumbens and other areas such as the prefrontal cortex.

Source: ONDCP, 1998c.

common link in the abuse and addiction of all substances. Dopamine has even been labeled "the master molecule of addiction" (Nash, 1997).

When the nucleus accumbens is functioning normally, communication among its neurons occurs in a consistent and predictable manner. First, an electrical signal within a stimulated neuron reaches its point of connection (i.e., the synapse) with the target neuron. The electrical signal in the presynaptic neuron triggers the release of dopamine into the synapse. The dopamine travels across the synaptic gap until it reaches the target neuron. It then binds to the postsynaptic neuron's dopamine-specific receptors, which in turn has an excitatory effect that generates an internal electrical signal within this neuron. However, not all of the released dopamine binds to the target neuron's receptors. Extra dopamine may be chemically deactivated, or it may be quickly reabsorbed by the releasing neuron through a system called the *dopamine reuptake transporter* (see Figure 2-4). As soon as the extra dopamine has been deactivated or reabsorbed, the two cells are "reset," with the releasing neuron prepared to send another chemical signal and the target neuron prepared to receive it. Substances of abuse, and especially stimulants, affect the normal functioning of the dopamine neurotransmitter system (Snyder, 1986; Cooper et al., 1991).

Figure 2-4
Dopamine's Normal Action



Note: After being released into the synapse (the gap between nerve endings and receiver cells), dopamine binds to receptors on the next neuron. The dopamine is either quickly reabsorbed or broken down by enzymes.

Neurological Reinforcement Systems

Psychologists have long recognized the importance of positive and negative reinforcement for learning and sustaining particular behaviors (Koob and LeMoal, 1997). Beginning in the late 1950s, scientists observed in animals that electrically stimulating certain areas of the brain led to changes in mental alertness and behavior. Rats and other laboratory animals could be taught to self-stimulate pleasure circuits in the brain until exhaustion. If stimulants such as cocaine or amphetamine were administered, for example, sensitivity to pleasurable responses was so enhanced that the animals would choose electrical stimulation of the pleasure centers in their brains over eating or other normally rewarding activities.

The process just described in which a pleasure-inducing action becomes repetitive is called positive reinforcement. Conversely, abrupt discontinuation of the psychoactive substances following chronic use was found to result in discomfort and behaviors consistent with craving. The motivation to use a substance in order to avoid discomfort is called negative reinforcement. Positive reinforcement is believed to be controlled by various neurotransmitter systems, whereas negative reinforcement is believed to be the result of adaptations produced by chronic use within the same neurotransmitter systems.

Experimental evidence from both animal and human studies supports the theory that stimulants and other commonly abused substances imitate, facilitate, or block the neurotransmitters involved in brain reinforcement systems (NIAAA, 1994). In fact, researchers have posited a common neural basis for the powerful rewarding effects of abused substances (for a review, see Restak, 1988). Natural reinforcers such as food, drink, and sex also activate reinforcement pathways in the brain, and it has been suggested that stimulants and other drugs act as chemical surrogates of the natural reinforcers. A key danger in this relationship, however, is that the pleasure produced by substances of abuse can be more powerfully rewarding than that produced by natural reinforcers (NIAAA, 1996).

Stimulants' Mechanisms of Action

On a short-term basis, stimulants exert their effects by disrupting or modifying the normal communication that occurs among brain neurons and brain circuits. Cocaine and MA have both been shown to specifically disrupt the dopamine neurotransmitter system. This disruption is accomplished by overstimulating the receptors on the postsynaptic neuron, either by increasing the amount of dopamine in the synapse through excessive presynaptic release or by inhibiting dopamine's pattern of reuptake or chemical breakdown (Cooper et al., 1991).

The use of cocaine and MA increases the amount of available dopamine in the brain, which leads to mood elevation (e.g., feelings of elation or euphoria) and increased motor activity. With cocaine, the effects are short-lived; with MA the duration of effect is much longer. As the stimulant level in the brain decreases, the dopamine levels subside to normal, and the pleasurable feelings dwindle away.

A growing body of scientific research based on animal research and brain imaging studies in humans suggests that the chronic use of stimulants affect dopaminergic neurons in limbic reward system structures (e.g., the VTA, nucleus accumbens). These effects may underlie addiction to stimulants. Although the neurochemical pathways of stimulant addiction are not definitively established, a few researchers have found evidence of changes in the structure and function of brain neurons after chronic stimulant use in humans. Some researchers propose that the changes may come from dopamine depletion, changes in neurotransmitter receptors or other structures, or changes in other brain messenger pathways that could cause the changes in mood, behavior, and cognitive function associated with chronic stimulant abuse (Self and Nestler, 1995).

Animal studies have demonstrated that high doses of stimulants can have permanent neurotoxic effects by damaging neuron cell-endings (e.g., Selden, 1991). The question of whether stimulants can produce similar effects in humans remains to be answered. Researchers hope that recently developed brain imaging techniques will help provide the answer. At this time, there is only speculation that such permanent damage may underlie the long-term cognitive impairments seen in some chronic stimulant users. The continuing development and application of new technologies will help expand our knowledge of the neurological effects of stimulants in humans. (The medical aspects of stimulant use disorders are discussed in Chapter 5.)

Abuse and Dependence

Addiction is a complex phenomenon with important psychological and social causes and consequences. However, at its core, it involves a biological process: the effects of repeated exposure to a biological agent (a substance) on a biological substrate (the brain) over time (Nestler and Aghajanian, 1997). Ultimately, adaptations that substance exposure elicits in individual neurons alter the functioning of those neurons, which in turn alters the functioning of the neural circuits in which those neurons operate. This eventually leads to the complex behaviors (e.g., dependence, tolerance, sensitization, craving) that characterize addiction (Koob, 1992; Kreek, 1996; Wise, 1996; Koob and LeMoal, 1997).

A general definition of *substance abuse* is the habitual use of a substance not needed for therapeutic purposes, such as solely to alter one's mood, affect, or state of consciousness. The continued abuse of the substance may lead to adverse physiological, behavioral, and social consequences. A substance-dependent individual will continue his use despite these adverse consequences. Moderate chronic use or severe short-term use of substances may lead to abuse, which may eventually lead to addiction components (Ellinwood, 1974; Hall et al., 1988; Kramer, 1969).

Chronic substance abuse results in a complex set of physiological and neurological adaptations. These adaptations are simply the body's attempt to adjust to or compensate for substance-induced impairments. Repeated exposure to a substance can also lead to adaptations in the reward circuitry that opposes and/or neutralizes a substance's effects (i.e., counteradaptation). *Substance addiction* (or *substance dependence*) is manifested by (1) psychological craving (see the following section); (2) tolerance (the need for increasing amounts of the substance to reproduce the initial level of response, or sometimes to simply stave off the unpleasant effects of withdrawal); (3) sensitization (discussed in the section on the medical effects of stimulants); and (4) symptoms of withdrawal upon cessation of use, indicating physiological dependence.

Social and behavioral manifestations of dependence include the reduced ability to function at work or home and may include displays of erratic, moody, or anxious behavior.

Similar to other substances of abuse, moderate chronic use or severe short-term use of stimulants in any form may lead to abuse or dependence (Ellinwood, 1974; Hall et al., 1988; Kramer, 1969). Clinical observations of abuse patterns for both cocaine and MA have noted that, in general, there is an estimated 2- to 5-year latency period between first use and full-blown addiction. However, clinical experience and anecdotal evidence suggest that the latency period may be shortened to less than 1 year by rapid routes of administration (e.g., injection, smoking) and increased stimulant purity (e.g., ice, crack). With increasing use, the user may develop tolerance to the effects of stimulants and may need to keep increasing the amount taken to produce the desired psychological effects. As chronic abuse progresses, users prefer the stimulant over enjoyable activities and eventually may prefer it over food and sex (Hall et al., 1988). At that point, the individual will usually continue her use even when faced with continuing adverse consequences—the hallmark of substance dependence. Abrupt discontinuation of the psychoactive substance following chronic use generally results in discomfort, dysphoria, and behaviors consistent with craving. The user is now motivated to use a substance in order to avoid discomfort and dysphoria. This shift from substance use as positive reinforcement to negative reinforcement is, perhaps, one of the foremost characteristics of late-stage addiction.

Drug Craving and Memory

The degree to which learning and memory sustain the addictive process has only recently been addressed. Researchers believe that each time a neurotransmitter like dopamine floods across a synapse, circuits that trigger thoughts and memories and that motivate action become hardwired in the brain. The neurochemistry supporting addiction is so powerful that people, objects, and places associated with substance use are also imprinted on the brain.

Craving, a central aspect of addiction, is a very strong learned response with powerful motivational properties often associated with specific memories (i.e., conditioned cues and triggers). Cues—any stimuli (substance-using friends, locations, paraphernalia, moods) repeatedly paired with substance use over the course of a client's addiction—can become so strongly associated with the substance's effects that the associated (conditioned) stimuli can later trigger arousal and an intense desire for the substance and lead to relapse. High relapse rates are common in cocaine addiction even after physical withdrawal and abstinence have been achieved.

Brain-imaging studies have shown that cue-induced drug craving may be linked to distinct brain systems involved in memory (e.g., London et al., 1990; Stapleton et al., 1995). Brain structures involved in memory and learning, including the dorsolateral prefrontal cortex, amygdala, and cerebellum, have been linked to cue-induced craving (Grant et al., 1995). A network of these brain regions integrates emotional and cognitive aspects of memory and triggers craving when it reacts to cues and memories. These cues and memories also play an important role in reinforcing substance use (Grant et al., 1995).

Most substance treatment programs recognize the power of these factors in triggering relapse and warn clients to avoid everything previously associated with their substance use—a tall order for a client in an urban environment saturated with the substance and its associated reminders. Treatment approaches that address these learning and memory issues of addiction may prove effective. For example, Childress developed treatment strategies to help clients reduce craving and arousal during encounters with substance-related stimuli (Childress, 1994). In the laboratory, clients are given repeated, passive exposure to substance-reminding cues in a substance-free protected environment. The research finds that initial arousal and strong craving produced by the cues eventually decrease (Childress, 1994). Better understanding of the relationship of learning and memory to the addiction process may lead to new treatment approaches.

Role of New Technologies

The recent development of noninvasive brain imaging has created a powerful new tool for demonstrating not only the short-term effects of substance use, but also the longer term consequences of chronic substance abuse and addiction. These tools have allowed researchers to boldly go where they previously could not—literally into the depths of a living human brain. Such noninvasive techniques can depict normal and abnormal functioning of different brain areas by measuring metabolic activity (i.e., glucose utilization). They can identify substance-induced structural changes and physiological adaptations. Through a combination of techniques, they can observe the altered "processing" of information in various circuits as the brain responds to substance use.

Using these techniques, investigators have been able to identify brain structures involved in craving, map the emotions of substance users, plot the neurobiological basis of substance-induced euphoria, and more. For example, researchers have used magnetic resonance imaging (MRI) and spectroscopy to see how brain structures change as substances produce their effects. Others have used a functional imaging technique called phosphorus magnetic resonance spectroscopy (^{31}P MRS) to show that chronic substance abuse is accompanied by abnormal metabolism in some areas of the brain that seems to return to normal when people stop using substances (Christensen et al., 1996). Positron emission tomography (PET) has revealed subtle alterations in the dopamine receptors of stimulant users' brains (Iyo et al., 1993). More recent PET studies have demonstrated long-term vulnerability to chronic stimulant abuse (Melega et al., 1997a; Volkow et al., 1996, 1997b). Another PET study has established a dose–response relationship between cocaine and the drug's subjective effects: The greater the amount of cocaine that is administered, the greater the high experienced by the user (Volkow et al., 1997a).

Other researchers combined electroencephalograms (EEGs) and MRI to produce a topographic brain map showing increased electrical activity (in the form of beta waves) during stimulant withdrawal (Herning et al., 1997). Mapping EEG activity during stimulant use and withdrawal may allow researchers to further document substance-induced neuropsychological impairments.

Although much is known about the effects of stimulants in animals, there is little such knowledge of these effects in humans (CSAT, 1997). The continuing development and application of new technologies such as noninvasive brain imaging will allow researchers to improve their understanding of how stimulants affect the human brain. Greater understanding of the underlying neuronal impairments of stimulant abuse will aid in the development of new, more effective treatment approaches.

General Effects Of Stimulants

Substances of abuse—and stimulants in particular—appear to increase the brain's levels of free dopamine in a dose-dependent manner; that is, more dopamine is available when higher doses of the substance are administered (Nash, 1997). The higher the substance dose, the greater the feelings of elation, euphoria, and satisfaction, and as the dopamine levels and pleasurable feeling subside, there is an intense desire to replicate the feelings of pleasure by administering another dose of the substance. This tendency toward repeated administration is characteristic of stimulant abuse and underlies most of the other effects of stimulants, as well as most other addictive substances.

Continued use often leads to adverse consequences, which may include neuropsychological impairment and diminished physical health. Work performance and social and family relations can be adversely affected, and the risk of arrest and conviction on drug-related charges increases. Even after a stimulant user discontinues use, impairments in cognition and functioning may persist, and there may even be persistent psychiatric symptoms (Wada and Fukui, 1990). Cravings for the stimulant's effects tend to linger, even after abstinence has been achieved, and the potential for relapse is high.

Medical Effects

Acute effects

The general acute effects of stimulants have been well documented. Among a range of physiological responses, stimulants are known to raise both systolic and diastolic blood pressure, increase heart rate, increase respiration rate, increase body temperature, cause pupillary dilation, heighten alertness, and increase motor activity (CSAT, 1997).

Acute effects from excessive doses include dangerously rapid and erratic heartbeat, cerebral hemorrhaging, seizures/convulsions, respiratory failure, stroke, heart failure, brain damage, coma, and death (CSAT, 1997).

Stimulants are also known to cause sensitization (i.e., the opposite of tolerance), for which multiple drug exposures eventually produce some new adverse reaction. For example, in animals, seizures do not typically occur after single low-to-moderate doses. But with repeated exposure, an animal can become sensitized to the stimulant and may have a seizure after a single, previously harmless, dose.

Chronic effects

Although the effects of chronic stimulant abuse in humans has not been well documented, some of the chronic effects include organ toxicity, compromised health (e.g., underfed; malnourished, poor hygiene), dental problems, and dermatitis. (For a complete discussion of the medical aspects of stimulant use, see Chapter 5.)

Psychological Effects

The immediate psychological effects of stimulant administration include a heightened sense of well-being, euphoria, excitement, heightened alertness, and increases in motor activity. Stimulants also reduce food intake, reduce sleep time, and may increase socialization activities. Stimulants may also enhance performance of certain types of psychomotor tasks.

High doses may result in restlessness and agitation, and excessive doses may produce stereotypic behaviors (repetitive and automatic acts). Chronic psychological effects of stimulant use include various psychiatric disorders such as psychosis, paranoia, and suicidal tendencies.

There may also be neurological impairments and cognitive deficits. Tolerance eventually develops to many of the behavioral effects of stimulants, so that increasing doses are required to achieve the same effect.

The administration of stimulants—particularly if smoked or injected intravenously—can have immediate and often very intense effects on the user. However, the "rush" and subsequent feelings of euphoria may just as quickly fade. The intense effects can be followed by a dysphoric "crash." To stave the crash, the user will administer another dose of the stimulant, which again produces a rush and subsequent crash.

This cycle will go on again and again. This pattern of frequently repeated dosing known as *bingeing* may continue for up to 3 sleepless days. During this period, the user may not eat and may lapse into a severe depression, followed by worsening paranoia, belligerence, and aggression—a period known as *tweaking*.

Bingeing eventually ends when the user depletes his supply of stimulants or simply collapses from sheer exhaustion. The stimulant user may then sleep for several days, only to awaken and begin the cycle again.

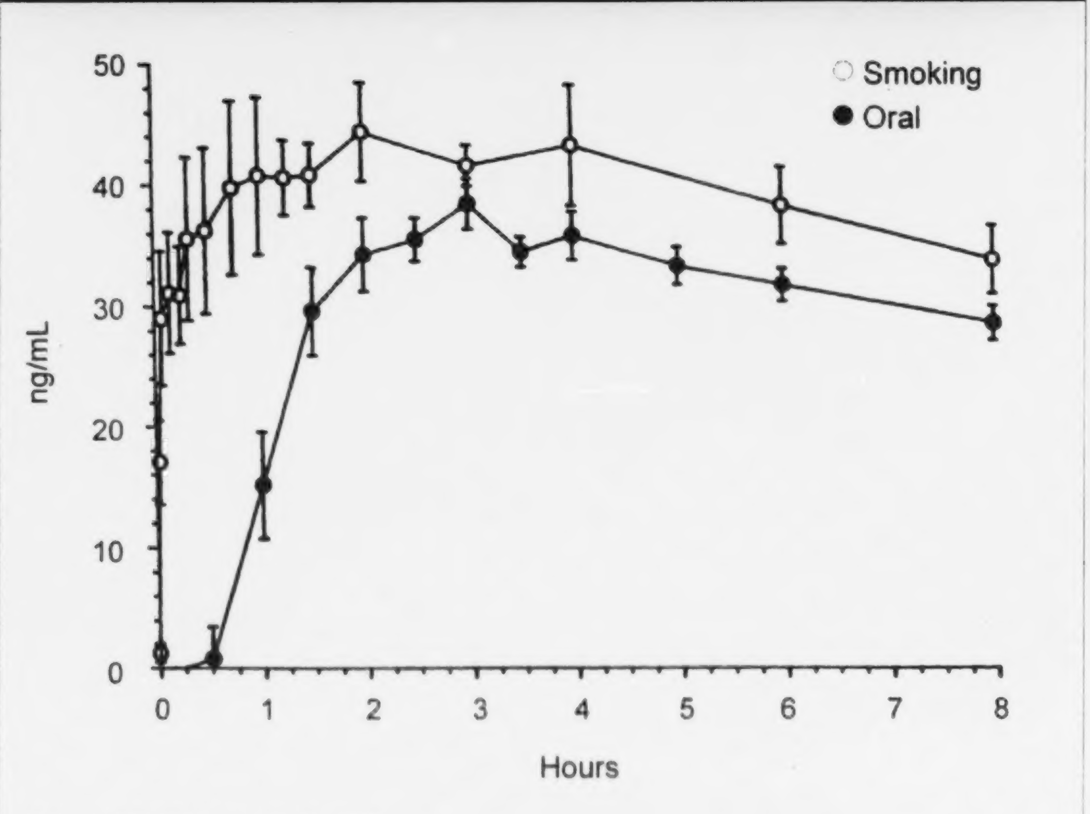
There is a great amount of anecdotal evidence on the relationship of stimulant use and various sexual behaviors. Stimulants may be used during sexual activities to intensify sexual acts, heighten pleasure, lengthen the duration of intercourse, and lessen inhibitions. The abuse of stimulants is also known to lead to uncharacteristically aberrant or deviant sexual behaviors, the use of prostitutes, and HIV high-risk behaviors (Rawson et al., 1998b).

Effects of Route of Administration

Cocaine and MA can be smoked, snorted, injected, or ingested orally. These various routes of administration differ in dosage and in the rapidity and intensity of effect, which may affect the course of abuse and dependence. Some evidence suggests that the onset of dependence varies according to the route of administration (DEA, 1995). The route of administration affects the amount (i.e., the dosage) of stimulant delivered to the brain, the speed at which it is delivered, and the resulting intensity of the stimulant's effects.

The intensity of the psychological effects of stimulants, as with most psychoactive drugs, depends on the dose and rate of entry to the brain. For example, when snorted, stimulants generally reach the brain within 3 to 5 minutes, and the resulting rush or "high" may not be perceived as immediate; intravenous administration produces a rush in about 15 to 30

Figure 2-5
Comparison of Plasma Levels of Methamphetamine After Oral Administration and Smoking



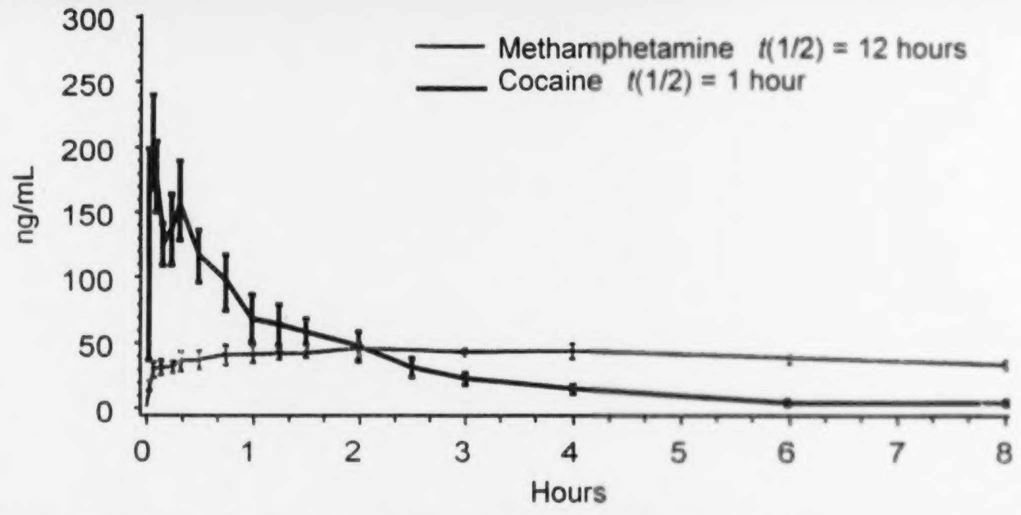
Source: Adapted from Cook, 1991.

seconds; whereas smoking produces an almost immediate effect (ONDCP, 1998a).

Because of the rapidity of delivery and higher dosages, the smoking of stimulants produces a high that is said to be far more intense than those produced through other routes of administration.

Route of administration has been shown to affect the resulting level of stimulant in the body. In a comparison of oral ingestion versus smoking, Cook measured plasma levels of MA after oral administration and after smoking (see Figure 2-5) (Cook, 1991). For the oral dose of 0.25 mg/kg, plasma levels began to rise 30 minutes after ingestion and reached peak levels (approximately 38 ng/mL) at about 3 hours after ingestion. Plateau levels were maintained until about hour 4 and then slowly declined over the next 4 hours. After smoking (dose of about 21 mg/subject), MA plasma levels approximated 80 percent of peak levels within minutes, peaked (approximately 42 ng/mL) at about 2 hours after administration, maintained a peak plateau for another 2 hours, and then slowly declined over the next 4 hours. By comparison, plasma levels of smoked cocaine and smoked MA both peaked rapidly (Cook, 1991). Plasma levels of smoked cocaine (dose of 21 to 22 mg/subject) peaked at approximately 240 ng/mL at about 5 to 10 minutes after administration. Cocaine plasma levels then declined rapidly, dropping to 50 percent of maximum level (half-life) within 1 hour. Smoked MA (dose of 21 to 22 mg/subject) neared peak levels (approximately 50 ng/mL) within minutes and continued to climb until about 2 hours after administration before slowly tapering off. However, half-life levels were not reached until 11 to 12 hours after administration (see Figure 2-6). The long plateau effect and the much longer half-life of MA versus cocaine suggests considerable dangers in repeated smoking of MA because remarkably higher plasma concentrations could be expected to occur if the dose is repeated, even at fairly long intervals (Cook, 1991). Because stimulants exert their effects in a dose-dependent manner, the route of administration has serious neurological, medical, psychiatric, and neurocognitive implications for the stimulant user and the treatment provider. The intense highs produced by smoking crack cocaine or ice MA can lead to equally intense "lows" during withdrawal (e.g., dysphoria, depression, irritability, anxiety, paranoia, dramatic mood swings). The subsequent cravings can also be extremely intense. Prolonged high doses of stimulants (e.g., during bingeing) may cause greater and longer lasting neurological damage, which in turn may lead to greater and longer lasting cognitive deficits. The onset of stimulants' chronic effects varies across individuals, and although there are few data to predict how long it will take for any user to begin suffering from the chronic effects of stimulant abuse and dependence, onset is probably related to the size of the doses, the frequency of dosing, and the route of administration. However, in general, the higher the doses and the more frequently the doses are administered, the more quickly the chronic effects will appear. From a treatment provider's perspective, a stimulant user's preferred route of administration affects the extent and depth of chronic effects and, therefore, has implications for choosing the most appropriate treatment approach. (See Chapter 4 for a full discussion on the practical applications

Figure 2-6
Comparison of Plasma Levels of Methamphetamine
And Cocaine After Smoking



Note: $t(1/2)$ = plasma half-life, which is the time it takes for the stimulant to drop to 50 percent of its peak level.

Source: Adapted from Cook, 1991.

of treatment strategies. For a discussion on route-of-administration effects on toxicity and adverse reactions, see Chapter 5.)

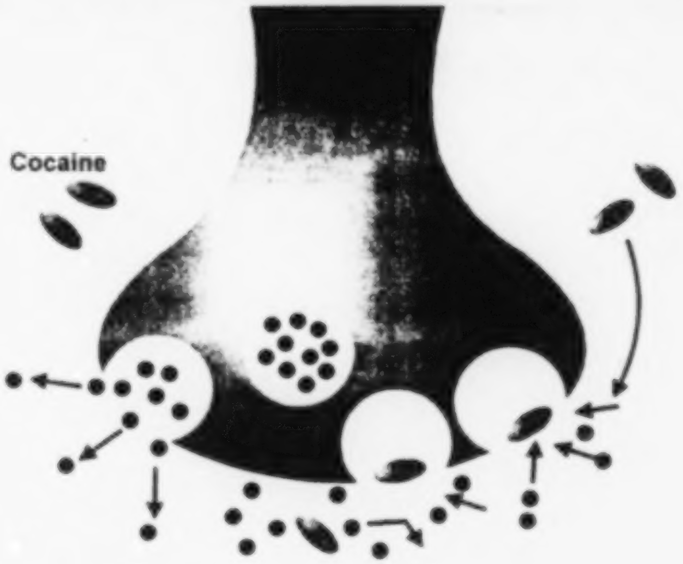
Cocaine

Acute Effects

Cocaine has two main pharmacological actions. It is both a local anesthetic and a central nervous system (CNS) stimulant—the only drug known to possess both of these properties. Cocaine exerts its local anesthetic actions by blocking the conduction of sensory impulses within nerve cells. This effect is most pronounced when cocaine is applied to the skin or to mucous membranes. Cocaine hydrochloride has approved medical use as a local anesthetic in surgery of the nose, throat, and larynx.

As a CNS stimulant, cocaine affects a number of neurotransmitter systems, but it is through its interaction with the dopamine and the limbic reward system that cocaine produces some of its most important effects, including its positive reinforcing effects. The major influence of cocaine on the dopamine system is its ability to block the synaptic reuptake of dopamine. As shown in Figure 2-7, cocaine does not directly "stimulate" the dopamine system; rather, it causes the system to be stimulated by preventing dopamine from being removed from the intracellular space. Cocaine blockade of the dopamine reuptake transporter extends the availability of dopamine in the synaptic space where it continues to occupy the dopamine receptor and causes the postsynaptic neurons to fire for a longer than normal period. This

Figure 2-7
Cocaine Blockade of the Dopamine Reuptake Transporter



Note: Cocaine blocks the normal reuptake of dopamine. As a result, dopamine accumulates in the synapse, where it continues to stimulate the receiver cell.

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extended firing of the postsynaptic neurons resulting from prolonged dopamine receptor activity is initially experienced subjectively by the cocaine user as a positive sensation involving increased energy, arousal, and stimulation. A recent study has demonstrated a relationship between the intensity of cocaine's subjective effects and the degree to which the dopamine reuptake transporter is blocked (Volkow et al., 1997a). The effects experienced by users of cocaine during the initial period of their use are generally mood-altering in a positive manner (Washton, 1989). For most individuals, the subjective experience of the acute effects includes a generalized state of euphoria in combination with feelings of increased energy, confidence, mental alertness, and sexual arousal.

Under the proper environmental circumstances, individuals also report that cocaine heightens their ability to concentrate, increases sexual excitement, increases their sociability, and decreases any preexisting shyness, tension, fatigue, depression, or boredom. Many people feel more talkative, more intensely involved in their interactions with others, and more playful and spontaneous when high on cocaine. As they come down from their cocaine high, some users experience temporary unpleasant reactions and aftereffects, which may include restlessness, anxiety, agitation, irritability, and insomnia. During this "rebound" period, suspiciousness, confusion, hyperarousal, and other elements of paranoid thinking may also appear.

With continued escalating use of cocaine, the user becomes progressively tolerant to the positive effects while the negative effects steadily intensify (Washton, 1989). Users report that the highs are not so high anymore and the rebound aftereffects increasingly lead to a dysphoric, depressed state. These new "lows" may reinitiate the desire for more cocaine in a futile attempt at mood normalization. The search for the previously experienced high will eventually leave the user in the depths of depression and despair.

When snorted, smoked, or injected intravenously, cocaine quickly produces an intense high. But because it is rapidly metabolized in the body, this high is short-lived. Efforts to replicate the initial high prompt users to take it often and repeatedly. Because of its mechanism of action, cocaine may produce strong craving and strong conditioning of cues associated with its use. The results of a recent brain imaging study revealed that cocaine's fast uptake in the brain has a major role in its rewarding effects and that its fast clearance from the brain sets the stage for frequent abuse, craving, and the binge pattern in cocaine addiction (Volkow et al., 1996). These researchers postulated that dopaminergic activation of the limbic reward system is involved in the rewarding effects of cocaine (and perhaps most, if not all, substances of abuse) and that continued activation of this system may lead to long-term changes in the associated neural circuits that perpetuate the compulsive administration of this drug (see below).

Cocaine use also has acute adverse physiological effects involving the respiratory, cardiovascular, and central nervous systems. Systemic toxicity to cocaine is characterized by profound sympathetic stimulation of the respiratory, cardiovascular, and central nervous systems, producing a combination of medical and psychological effects sometimes known as the "cocaine reaction." (For additional details on the medical aspects of cocaine abuse, see Chapter 5.)

Chronic Effects

For many cocaine users, the initial experimental use begins to give way to more frequent or regular use. With continued, intensified use, the "casual" user will progress to the abuse stage, requiring larger and larger doses to achieve the desired effects. The abuser may become obsessed with the rituals of cocaine administration and may find that many common items or situations trigger cravings for the drug. For some, abuse will lead to full-fledged addiction. There will be overwhelming urges and cravings for cocaine, and there may be an inability to self-limit or control use. The cocaine addict will deny that she has a drug use problem and will continue use of cocaine despite the negative consequences. At this stage, the adverse consequences of cocaine addiction have probably affected all aspects of the user's life. The addict has succumbed to what Dr. Sidney Cohen called cocaine's "pharmacological imperative" (Washton, 1989). Figure 2-8 lists the characteristics of the stages of cocaine addiction.

Figure 2-8

The Course of Cocaine Addiction

Early Stage

- Brain chemistry altered
- Addictive thinking begins
- Obsessive thoughts
- Compulsive urges
- Conditioned cravings
- Lifestyle changes
- Withdrawal from normal activities
- Subtle physical and psychological consequences (e.g., jitters, irritability, mood swings)

Middle Stage

- Loss of control
- Cravings
- Inability to stop despite consequences
- Denial
- Increasing isolation
- Increasing physical and psychological consequences (e.g., paranoia, panic seizures)
- Impaired work/school performance

Late Stage

- Failure of efforts to stop
- Severe financial problems
- Severe work/school dysfunction
- Plummeting self-esteem
- Severe relationship problems
- Chronic severe depression
- Cocaine psychosis
- Death

Source: Washton, 1989. Copyright © 1989 by Arnold Washton. Used with permission from the author.

The timetable for the onset of the chronic effects of cocaine use varies across individuals and may depend on the size of the doses, the frequency of dosing, and the route of administration. There are no data to base a prediction on how long it will take for any individual to begin to suffer from the chronic effects of cocaine use. However, similar to the effects of MA, the higher the doses and the more frequently the doses are administered, in general, the more quickly the chronic effects of cocaine will appear. In addition, intranasal administration (snorting) is associated with slower onset of chronic effects than if cocaine is smoked (freebase or crack) or injected intravenously. Clearly, there are tremendous individual differences in this timetable, with some individuals reporting an ability to use for extended periods with few signs of negative consequences and others reporting a very dramatic onset of severe detrimental effects as soon as a few weeks or months after initiation of cocaine use.

Physically, the cocaine addict may appear thin or even emaciated. Personal hygiene and self-care may be neglected, and medical and dental needs may go unmet. Because cocaine suppresses appetite, the user fails to eat properly and may suffer from vitamin deficiencies. Severe addicts may ignore food, clothing, shelter, and sexual needs.

Psychologically, cocaine's chronic effects are exactly the opposite of the desired initial effects. Continued cocaine use increases paranoia and confusion and causes an inability to concentrate and an inability to perform sexually. The same substance that acutely produced a mild sensation of arousal and decreased fatigue, on a chronic basis results in chest pain, insomnia, anorexia, episodic depression, and extreme fatigue.

From a treatment perspective, the curious thing is that the user often accurately perceives and attributes the pleasurable, acute effects to the use of cocaine. However, he frequently is unable or unwilling to recognize the relationship of the negative, chronic effects to the use of cocaine. Although it may be dramatically apparent to family and friends that the effects of cocaine are highly detrimental and destructive to the user, the user may insist that the use of cocaine is very helpful and beneficial. The extensive health-compromising effects of cocaine abuse are apparent when examining the behavioral and psychological profile of clients as they enter substance treatment. Generally, these clients exhibit a pronounced disruption in healthy behaviors and an elevation in dysphoric emotions including anxiety, depression, and paranoia (Castro et al., 1992).

Chronic abuse of cocaine may cause neuropsychological impairments (O'Malley et al., 1992) as well as neuropsychiatric syndromes (Herning et al., 1997). Cocaine-induced cognitive deficits can last up to 3 months after heavy use before baseline functioning is restored. In their review, Weinrieb and O'Brien found a strong association between the chronic use of cocaine and deficiencies in short-term auditory recall, memory, concentration—especially for nonverbal abstracting and problem solving—and slowed reaction time (Weinrieb and O'Brien, 1993).

The physical, psychological, and cognitive effects of chronic cocaine use reflect the underlying physiological effects; at the heart of these effects is cocaine's impact on the neurotransmitter dopamine.

Methamphetamine

Acute Effects

Although research efforts continue to focus on the effects of MA, there are limited data on MA's effects on humans (CSAT, 1997). Much of the available information has been surmised from the literature on cocaine. However, the physiological effects of MA are generally similar

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to those of cocaine: increased heart rate, elevated blood pressure, elevated body temperature, increased respiratory rate, and pupillary dilation. Other acute effects include rapid heart rate, irregular heart rate, and irreversible, stroke-producing damage to small blood vessels in the brain.

MA's psychological effects, like those of cocaine, include a heightened sense of well-being or euphoria, increased alertness, increased vigor, decreased food intake, and decreased sleep time. Acute administration has been shown to increase socialization in humans. High doses may produce repetitive and automatic acts in both humans and animals, and in humans, may cause irritability, aggressive behavior, excitement, auditory hallucinations, and paranoia (delusions and psychosis). Dangerously elevated body temperature and convulsions occur with MA overdoses, and if not treated immediately, can result in death. With continued use, tolerance develops to the behavioral effects, and repeated exposure may produce sensitization. MA users tend to engage in violent behavior. Mood changes are common, with the user rapidly changing from friendly to hostile.

The course of addiction to MA is believed to be similar to that of cocaine. Even the underlying neurological effects of MA are similar to the effects produced by cocaine: increased levels of free dopamine in the brain's limbic reward system. The MA "withdrawal syndrome" is like that of cocaine, but due to the longer effects of MA, withdrawal may be more intense and protracted. Several hours after last use, the MA user experiences a drastic drop in mood and energy levels. Sleep—which may be promoted by the use of secondary substances such as alcohol, barbiturates, and benzodiazepines—finally begins and may last for several days. Upon awakening, the user may experience severe depression, perhaps lasting for several weeks. While in this depressed state, the user has an increased risk of suicide. But once the user feels that she "has recovered" from a bingeing episode, cravings set in, and the cycle often begins again.

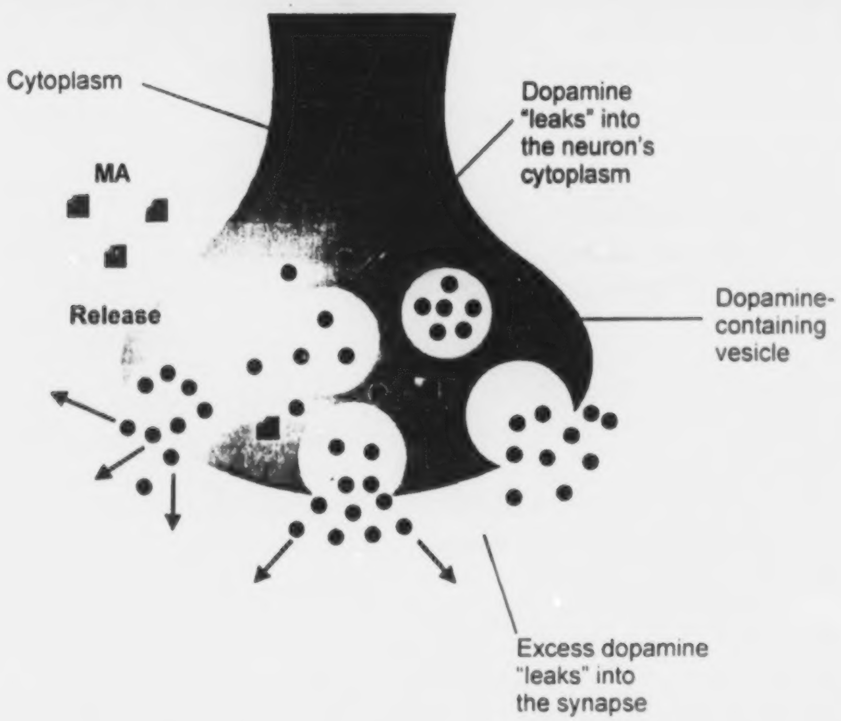
There are three essential differences between cocaine and MA. First, MA is thought to enhance CNS neurotransmission by increasing the presynaptic release of dopamine within the limbic reward system. Second, recent research has demonstrated MA's neurotoxicological effects in animals and has begun to support the hypothesis that MA is neurotoxic in humans. Unlike cocaine, MA does cross neuronal cell membranes and will enter into the microscopic sacs (called *vesicles*) where neurons store dopamine. MA is believed to damage the storage sacs and the neurons' axonal endings such that dopamine leaks uncontrollably into the synapse (see Figure 2-9). MA can also cause neurotoxicity indirectly by mobilizing dopamine out of the safe storage vesicles within the neuron and into the neuron's cytoplasm (i.e., the cell's internal material) where it is converted to toxic and reactive chemicals. Third, cocaine is rapidly metabolized by plasma and tissue enzymes, whereas MA is metabolized at a much slower rate, which results in a longer duration of action (Cook, 1991; ONCDP, 1998b). Although the half-life (effective duration of action) of cocaine is 1 to 2 hours, a single dose of MA may produce an effect for 8 to 12 hours. The fact that MA is metabolized at a slower rate also allows more time for MA to exert its neurotoxicological effects.

The sustained high plasma levels suggest considerable dangers in repeated smoking of MA because remarkably higher plasma concentrations could be expected to occur if the dose is repeated, even at fairly long intervals (Cook, 1991).

Chronic Effects

Chronic abuse of MA may result in inflammation of the heart lining and, among

Figure 2-9
Methamphetamine's Effects on Synaptic and Intraneuronal Dopamine Leakage


Cytoplasm
MA
Release
Dopamine "leaks" into the neuron's cytoplasm
Dopamine-containing vesicle
Excess dopamine "leaks" into the synapse

Note: Methamphetamine causes excess release of dopamine, overwhelming the processes of reuptake and enzyme breakdown. Methamphetamine also causes dopamine to leak out of the vesicles.

users who inject the drug, damaged blood vessels and skin abscesses. Chronic users may also have episodes of violent behavior, paranoia, anxiety, confusion, and insomnia. Heavy users show progressive social and occupational deterioration. Psychotic symptoms may sometimes persist for months or years after use has ceased.

Some of the most frightening research findings about MA suggest that its prolonged use not only modifies behaviors, but literally changes the brain in fundamental and long-lasting ways. Animal studies have shown that chronic use of MA can significantly reduce brain dopamine levels for up to 6 months after last use, with less significant reductions persisting for up to 4 years. MA impairs the functioning of both the dopamine system and the serotonin system (serotonin is another important CNS neurotransmitter). MA-induced neuronal toxicity is specific to certain brain regions (primarily the limbic reward system), and this toxicity is reflected both biochemically and anatomically. The adverse effects produced by MA are often long-lasting, and there is some speculation that some types of damage may be permanent. Finally, these impairments in brain functioning may underlie the cognitive and emotional deficits seen in many MA users. Understanding the chronic effects of MA use is essential for treatment providers who serve this population.

Animal studies have shown that high dose regimens of MA significantly deplete neurotransmitter levels, particularly those of dopamine (e.g., Seiden et al., 1976). Subsequent studies replicated these findings (e.g., Ricaurte et al., 1980) and demonstrated that these depletions were evident up to 4 years after cessation of MA administration (Woolverton et al., 1989). A more recent study demonstrated that chronic amphetamine exposure in monkeys could produce long-term effects on the brain's ability to produce dopamine (Melega et al., 1997a). Significant depletion of dopamine persisted 6 months later; even after 1 year, the brain dopamine levels were only at 80 percent of their preexposure levels. In a radiotracer study of humans, Iyo and colleagues (Iyo et al., 1993) revealed reductions in dopamine receptor binding availability in brain areas such as the frontal cortex and striatum in MA users. Although there is little current evidence on MA's chronic effects in humans, animal research has proven that prolonged or heavy use of MA dramatically reduces the brain's ability to produce dopamine.

Numerous animal studies have demonstrated that MA can damage both dopamine and serotonin systems (e.g., Peat et al., 1983; Robinson and Becker, 1986; Seiden et al., 1976; Trulson and Trulson, 1982a, 1982b; Wagner et al., 1979). MA toxicity occurs after repeated high-dose administration, and it is selective for certain neuronal systems, particularly those in the limbic reward system (e.g., striatum, substantia nigra, nucleus accumbens). Within these brain circuits, MA has been shown to reduce the number of nerve fibers, impair normal physiological functioning, and destroy both axons and axon terminals (i.e., at synaptic junctions). These studies have also shown that MA toxicity is highly dependent on dose, route of administration, and frequency with which the drug is given.

Prolonged or heavy use of MA decreases the brain's ability to manufacture dopamine. This impairment may persist for up to 1 year after the user has stopped taking MA. Researchers now believe that those changes in dopamine and the damage done to dopamine and serotonin neurons are responsible for the chronic effects of MA use that are much more pronounced than the acute effects.

If MA does indeed cause damage to dopamine and serotonin systems in humans, then there are ramifications to consider. One of the outcomes of chronic MA use is psychosis. Psychotic individuals are often treated with drugs to reverse or return their brain functions to normal, and most antipsychotic medications work by changing the activities of the dopamine and serotonin neurons. The unanswered question is: Will antipsychotic medications be able to effectively treat MA-induced psychoses in individuals whose dopamine and serotonin systems have been impaired by chronic MA abuse? To date, there have been few, if any, studies investigating antipsychotic medications for the treatment of chronic MA abuse and dependence.

In summary, although there is much evidence of MA's neurotoxicity in animals, the issue of whether MA causes permanent damage to dopamine and serotonin neurons in humans remains very much an unanswered question. Because of the inherent dangers associated with this type of research, the information will have to come from postmortem studies, advanced neuroimaging studies, and the development of new strategies for detecting neurotoxicity—possibly through the use of operant behavioral pharmacology. Finally, the degree of neurotoxicity must be placed in perspective, and the functional consequences require further scrutiny to determine the impact of chronic MA abuse on human brain function.

Summary

Recent research has shown how stimulants such as cocaine and MA exert their effects on the user's nervous system and change the user's feelings, emotions, and behavior. There is now a greater understanding of neurological reinforcement systems, how substance use can lead to dependence, and the roles that craving and memory play in sustaining addiction. Although there is currently a dearth of research regarding the neurologic, medical, psychiatric, and neurocognitive effects of stimulants in humans (CSAT, 1994b, 1997), animal studies have demonstrated cocaine's and MA's ability to disrupt normal brain function and cause long-lasting and perhaps permanent neurological impairments. With continuing research and the development of new imaging technologies, the full extent of these stimulants' effects on humans will eventually be revealed. This new information should continue to assist in the development of new and improved approaches for treating stimulant use disorders.

3 Approaches to Treatment

As large numbers of people with substance use disorders began to seek treatment in the early and mid-1980s, "treatment" for stimulant abuse and dependence was invented. The treatment system that responded most quickly was the 28-day Minnesota Model hospital industry. The number of these 28-day, for-profit treatment units grew at an astonishing rate. Tens of thousands of cocaine users were treated in these programs with strategies adapted from the treatment of alcoholics. Today, there is little empirical evidence to assess the efficacy of these efforts.

During this same period, all sorts of unconventional remedies, including health foods, amino acids, hot tubs, electronic brain tuners, and other "New Age" treatments emerged and disappeared. Research efforts to develop scientifically based treatments began during this period with behavioral techniques like contingency contracting (Anker and Crowley, 1982) and medication evaluations including the use of desipramine (Norpramine) (Tennant and Rawson, 1983; Gawin and Kleber, 1984). Over the 15-year period since these early efforts, an entire stimulant use disorder treatment literature has developed.

This chapter reviews the current state of knowledge on the treatment of stimulant use disorders, beginning with the approaches that have the most rigorous empirical support. Other approaches with less support in the scientific literature are presented later in the chapter. At the end of the chapter is a review of the current state of medications research in the treatment of stimulant use disorders. Although at the time of this writing there were no medications with demonstrated clinical efficacy, the ongoing program of research sponsored by the National Institute on Drug Abuse (NIDA) holds great promise for important treatment advances. For this reason, the current state of this research effort will be reviewed.

Documented Treatment Approaches

How To Measure Effectiveness

This chapter reviews what is scientifically known about effective treatments for stimulant use disorders. To be judged effective, a treatment must have been tested and demonstrated to be effective in a randomized clinical trial. Many psychosocial and pharmacological treatments have been investigated in such trials. Several psychosocial treatments for stimulant abuse and dependence have been found to be effective, but to date, no reliably effective pharmacological treatments have been found. What has been learned so far about the use of psychosocial and pharmacological treatments for stimulant use is summarized below. Almost all of the information has been gleaned from studies conducted with cocaine users. Similar studies with methamphetamine (MA) users have not been reported. However, evidence from at least one study indicates that cocaine and MA users respond similarly to psychosocial interventions, suggesting that what has been learned from cocaine users may be applicable to MA users (Huber et al., 1997).

Randomized clinical trials are the best available method for determining whether an intervention improves health. A randomized clinical trial is a prospective study comparing the effect of some intervention against a control intervention in groups of clients who are assigned randomly to the respective treatment groups (see Friedman et al., 1983). In such trials, clients from a particular population sample (e.g., all admissions to clinic X during 1998 meeting a particular list of inclusion and exclusion criteria) are randomly assigned to the intervention under study or to a control condition. Random assignment ensures against possible bias in assigning particular kinds of clients to the respective groups and helps to distribute evenly between the groups any subject characteristics that might influence outcomes.

Prospective means that clients in the groups are studied from the start of the intervention as opposed to retrospectively compiling the information after the intervention is completed. Retrospective observations tend to be less accurate because of relevant information not being collected, getting lost, or being distorted through reliance on people's recall. Having a comparison or control group is essential because most problems have some level of variability (i.e., they wax and wane over time) and because many health problems resolve over time without any formal treatment. The most effective way to determine whether any observed changes are due to the treatment being investigated rather than natural variability is by comparing against a similar group of clients who either received no treatment or received a standard treatment.

Some of the alternatives to randomized clinical trials common in the substance use disorder treatment field can provide useful information but have serious limitations that must be recognized. For example, following a group of clients who received a particular treatment in the absence of a comparison group can be informative in terms of characterizing what has happened to them (e.g., percentage relapsed, percentage who received additional treatment, amount of change from pre- to posttreatment), but such observations do not permit any scientifically valid inferences regarding the role of the treatment provided to any of the changes observed during followup. For that purpose, a comparison group is necessary. Any changes observed might have occurred in the absence of treatment. Without a comparison group there simply is no way to rule out that possibility. Similarly, when clients themselves select group membership, as opposed to being assigned by the researcher, one cannot make valid inferences about the role of treatment to outcome. For example, comparing treatment completers to dropouts is common and may be informative in terms of characterizing how the groups fared, but it is not scientifically valid to infer that any differences observed between them were due to the different amounts of treatment received. It very well could be that some other factor (e.g., differences in the amount of other demands on their time) was responsible both for the differential retention rates and for the subsequent differences observed at followup.

Psychosocial Treatment Approaches

The psychosocial interventions demonstrated thus far to be efficacious in randomized clinical trials with stimulant users share a common feature of incorporating well established psychological principles of learning.

It is impossible to quantify all aspects of psychosocial treatment. Often therapists working in the same clinic and using the same treatment approach differ greatly in terms of the progress their clients make. Put simply, some therapists appear to be very effective and others relatively ineffective. The use of carefully prepared treatment manuals reduces such between-therapist differences. Treatment manuals increase the likelihood that therapists will deliver a uniform set of services to their clients. That does not come at the cost of eliminating therapists' clinical judgment or flexibility. A carefully prepared manual recognizes the importance of clinical judgment and flexibility in addressing the individual needs of clients and incorporates those features into the manual. Considering that effective treatments and associated manuals are available, using them is prudent and will help ensure that clients receive the services that research has shown to be effective.

Community-Reinforcement-Plus-Vouchers Approach

Community reinforcement is an individualized treatment designed to promote lifestyle changes in several key areas that are conducive to successful recovery (see Meyers and Smith, 1995; Sisson and Azrin, 1989). First, clients with spouses who are not themselves users are offered marital therapy to improve the quality of their relationships in a reciprocal and rewarding manner. Second, clients who are unemployed, employed in jobs that are high-risk for substance abuse, or need vocational assistance for some other reason receive help in that domain. Third, clients are counseled and assisted in developing new social networks and recreational practices that promote and support recovery. Self-help participation is not mandatory but is often used as an effective means of developing a new social network. Fourth, various types of skills training are provided depending on individualized client needs, including substance refusal and associated skills, social skills, time management, and mood regulation training. Finally, clients with alcohol use disorders and no medical contraindications are offered a program of disulfiram (Antabuse) therapy coupled with strategies to support medication compliance.

Voucher-based incentive programs are designed to facilitate retention in treatment and to promote initial abstinence from stimulants. Such incentive programs are known as contingency management interventions, which are discussed further below. In this treatment, clients earn vouchers that are exchangeable for retail items contingent on stimulant-free urinalysis results during the initial 12 weeks of the 24-week treatment. Urinalysis monitoring is conducted thrice weekly during that period. The voucher system used in studies evaluating this treatment included incentives worth a maximum of approximately $980 across the course of treatment. Since those studies were completed, others have reported effective voucher programs using lower cost incentives (Tusel et al., 1995); another program obtained all its incentives via donations from community businesses (Amass, 1997), although the efficacy of this program was not evaluated. How valuable the incentives must be to significantly improve outcomes has not yet been evaluated.

The efficacy of the community-reinforcement-plus-vouchers approach, delivered as a comprehensive, stand-alone treatment, is supported by three randomized clinical trials (Higgins et al., 1993b, 1994b, 1997), with several additional trials supporting the efficacy of particular components of that approach (e.g., Silverman et al., 1996). The first trial examined the efficacy of this treatment compared with standard outpatient counseling (Higgins et al., 1993b). Treatment was 24 weeks in duration with 6 months of additional followup. The community-reinforcement-plus-vouchers treatment retained clients significantly longer and documented significantly longer periods of continuous stimulant abstinence than did standard counseling. For example, 58 percent of clients assigned to the community-reinforcement-plus-vouchers treatment completed 24 weeks of treatment compared with 11 percent of those assigned to standard counseling. Furthermore, of the clients in the community-reinforcement-plus-vouchers group, 68 percent were documented to have achieved 8 weeks of continuous cocaine abstinence, and 42 percent had 16 weeks of continuous abstinence. Of the clients in the standard counseling group, only 11 percent were documented to have achieved 8 weeks of continuous cocaine abstinence, and only 5 percent had achieved 16 weeks of continuous abstinence. Followup assessments revealed another important difference: Greater cocaine abstinence was documented—at 6, 9, and 12 months after treatment entry—in the group that received community-reinforcement-plus-vouchers treatment than in those who received standard counseling (Higgins et al., 1995).

A detailed manual (Budney and Higgins, 1998) that was designed specifically to guide clinicians in the day-to-day implementation of this approach was published recently by NIDA and is available at no cost via the NIDA Clearinghouse (1-800-729-6686) or can be downloaded from the website http://www.nida.nih.gov/TXManuals/CRA/CRA1.html

Contingency Management

The voucher system mentioned above is a contingency management intervention (also referred to as contingency contracting). Contingency management is a well-known behavioral intervention that is designed to increase or decrease desired behaviors by providing immediate reinforcing or punishing consequences when the target behavior occurs. Contingency management has been used with considerable effectiveness in the treatment of a variety of types of substance use disorders and is very useful for treatment planning because it sets concrete short-term and long-term goals and emphasizes positive behavioral changes (Stitzer and Higgins, 1995). However, relying exclusively on punitive consequences in contingency management interventions is not recommended because doing so can promote early treatment dropout (Stitzer et al., 1986).

The voucher program has been demonstrated to be efficacious when delivered apart from the community reinforcement treatment. Silverman and colleagues, for example, demonstrated that vouchers contingent on cocaine-negative urinalysis results increase cocaine abstinence in methadone maintenance clients who abuse cocaine (Silverman et al., 1996). Tusel and colleagues demonstrated reductions in all illicit substance abuse with contingent vouchers (Tusel et al., 1995).

Although vouchers are a well-supported contingency management intervention for increasing abstinence in stimulant users, other methods are also effective. Examples among methadone maintenance clients are take-home methadone doses (which eliminate the need for methadone clients to visit the clinic daily to consume their medication under staff supervision) (Stitzer et al., 1992), continuance of methadone maintenance treatment contingent on abstinence from cocaine (Kidorf and Stitzer, 1993), and even a simple system wherein publicly displayed gold stars and inexpensive gifts (e.g., coffee cups, gasoline coupons) are earned for substance abstinence and counseling attendance (Rowan-Szal et al., 1994).

Contingent methadone take-home doses have been used effectively when coupled with other treatment services. An excellent example of this was provided by McLellan and colleagues (McLellan et al., 1993). Methadone maintenance clients were randomly assigned to one of three conditions that provided increasing levels of services. Two of the three groups received methadone take-home doses contingent on negative urinalysis results and proof of current employment. These groups also received additional services not provided to the minimal-service group. The two groups given the opportunity to earn contingent take-home methadone doses achieved higher rates of cocaine and opiate abstinence than did clients receiving noncontingent take-home doses.

Iguchi and colleagues investigated whether cocaine abstinence could be increased through contingent reinforcement of compliance with individualized treatment plans rather than negative urinalysis results (Iguchi et al., 1997). Newly admitted methadone maintenance clients were assigned to one of three groups: (1) a control group receiving standard treatment at the methadone clinic (the standard group); (2) a group receiving standard treatment plus monetary vouchers contingent on the submission of substance-free urine specimens (urinalysis-contingent group), or (3) a group receiving standard treatment plus the same monetary vouchers but contingent on completing treatment plan tasks (treatment plan group). The third group demonstrated significantly greater reductions in illicit substance use than did the other two groups.

Contingency management can be effective with more-difficult-to-treat subgroups of stimulant users. For example, a contingency management approach that was efficacious in homeless stimulant users combined nonhospital day treatment with access to work therapy and housing contingent on substance abstinence (Milby et al., 1996). Nearly three-fourths of the subjects in this study were primarily crack cocaine users. They were randomly assigned to receive either enhanced or usual care. Enhanced care consisted of 2 months of clinic attendance for 5.5 hours each weekday, transportation to and from the clinic, lunch, psychoeducational groups, and individualized counseling.

During the last 4 months of the trial, the intensity of day treatment was reduced to allow subjects to participate in a work-therapy program refurbishing condemned houses in which they could live for a modest rental fee. Participation in the work program and housing were contingent on the provision of weekly random urinalysis testing. Drug-positive results precluded subjects from working in the program and required them to vacate the housing within 2 weeks. The work and living arrangements could be resumed on submission of two consecutive substance-free urine specimens. Usual care consisted of twice-weekly, 12-Step-oriented group and individual counseling, medical evaluation and treatment or referral, and referrals to community agencies for housing and vocational services. Enhanced care increased cocaine abstinence significantly at the 2-month assessment, although not at the 6- or 12-month assessments. Enhanced care also produced greater reductions in alcohol use at each assessment and significantly fewer days homeless at the 6- and 12-month assessments.

Pregnant women are another important subgroup with whom contingency management has been evaluated, although only in the form of preliminary studies. In two pilot studies, pregnant women were offered incentives for attendance at prenatal clinics and/or maintaining cocaine abstinence (Elk, in press). Monetary vouchers of increasing value were awarded for each successive substance-free urine specimen and for increased or consistent attendance at prenatal and substance use disorder treatment clinics. Abstinence, retention rates, and compliance with prenatal care visits were generally higher in the contingency groups. In another study, pregnant clients were randomly assigned to receive standard or enhanced methadone maintenance treatment (Carroll et al., 1995a). Standard treatment consisted of daily methadone, weekly group counseling, and thrice-weekly urine testing. Enhanced treatment consisted of weekly prenatal care, weekly relapse prevention groups, and monetary vouchers for every three consecutive substance-free urine samples. Treatment retention was similar in the two groups, and there were no significant differences in the percentage of cocaine-positive urine samples provided by the two groups.

This treatment approach with pregnant women with stimulant use disorders is very preliminary and needs more thorough evaluation. However, these efforts further illustrate the potential utility of contingency management for addressing some of the more daunting clinical challenges in treating stimulant abuse. Other important examples are recent pilot studies (Roll et al., 1998; Shaner et al., 1997) suggesting that contingent monetary reinforcement can reduce cigarette and cocaine use in adult schizophrenic clients and providing evidence that contingent monetary reinforcement can be used to increase medication compliance in tuberculosis-infected stimulant users (Elk, in press).

When considered as a group, contingency management interventions have by far the greatest amount of empirical support for their efficacy in promoting therapeutic behavioral change among stimulant users. Stimulant users are sensitive to systematically applied contingency management interventions. Presently, there is no other treatment strategy about which one can make an equally strong positive statement.

Relapse Prevention

Relapse prevention (RP) systematically teaches clients (1) how to cope with substance craving, (2) substance refusal and assertiveness skills, (3) how seemingly irrelevant decisions can affect the probability of later substance use, (4) general coping and problem solving skills, and (5) how to apply strategies to prevent a full-blown relapse should an episode of substance use occur (Marlatt and Gordon, 1985).

Carroll and colleagues have adapted and demonstrated the efficacy of this treatment approach with cocaine users (Carroll et al., 1991a, 1991b, 1994a, 1994b). In an initial study, RP was compared with interpersonal psychotherapy (IP), which teaches strategies for improving social and interpersonal problems (Carroll et al., 1991a). Retention was better with RP than IP, and trends suggested cocaine abstinence may have been as well, but that difference was not significant.

A subsequent study compared RP and case management (Carroll et al., 1994a); the clients in this study also received either desipramine or placebo. A total of 139 clients were randomized to one of four treatment groups. Case management was designed to provide a nonspecific therapeutic relationship and an opportunity to monitor clients' clinical status. Both treatments were delivered in weekly therapy sessions during 12 weeks of treatment. All clients also received weekly urinalysis testing and other clinical monitoring. All treatment groups improved from pre- to posttreatment on measures of cocaine use and the Addiction Severity Index (ASI) drug, alcohol, family/social, and psychiatric composite scales, but there were no significant main effects for psychosocial (RP vs. case management) or drug treatment (desipramine vs. placebo). At 1-year followup, those clients who received RP reported significantly higher levels of cocaine abstinence than did clients who received case management (Carroll et al., 1994b). Considering RP's focus on teaching skills to prevent a lapse from becoming a full-blown relapse, these delayed effects might be expected. Indeed, similar delayed effects of RP have been reported in studies on treatment of other types of substance use disorders (see Carroll, 1996).

Not all studies with RP have been positive. For example, Wells and colleagues reported negative results in a comparison of RP and 12-Step-based counseling (Wells et al., 1994). No significant differences between the two groups were discerned in retention or cocaine use during the 24-week outpatient trial or at a 6-month followup evaluation.

Treatment Approaches With Supportive Research

The Matrix Model

The Matrix model (originally referred to as the neurobehavioral model) is an outpatient treatment approach that was developed during the mid-1980s for the treatment of individuals with cocaine and MA use disorders (Rawson et al., 1990). The model integrates treatment elements from a number of specific strategies, including relapse prevention, motivational interviewing, psychoeducation, family therapy, and 12-Step program involvement. The basic elements of the approach consist of a collection of group sessions (early recovery skills, relapse prevention, family education, and social support) and 20 individual sessions, along with encouragement to participate in 12-Step activities, delivered over a 24-week intensive treatment period (Rawson et al., 1989).

This treatment model serves as the primary treatment protocol for a network of outpatient treatment offices in Southern California (Matrix Center). In this network of clinics, more than 8,000 people with cocaine and MA use disorders have been treated with this approach since 1985. The client population ranges from professionals and executives to inner-city crack users and indigent rural MA users. In order to adapt to the financial realities imposed by the emergence of managed care, 2-month and 4-month versions of the model have been developed and are currently being evaluated. As the model was developed and refined, an extensive set of data on the value of the treatment approach was collected. The research studies evaluating this treatment approach do not include a randomized clinical trial. However, in seven research projects evaluating the treatment model, application of the model has been shown to be associated with significant reductions in cocaine, MA, and other substance use (Rawson et al., 1993, 1996; Shoptaw et al., 1994). In a project comparing the treatment outcome of 224 cocaine and 500 MA users to the Matrix approach, all indicators suggested a comparable treatment response (Rawson et al., 1996; Huber et al., 1997). Along with a reduction of stimulant and other substance use, treatment participation in the Matrix model has been demonstrated to be associated with a significant reduction in HIV-risky sexual behavior (Shoptaw et al., 1997). See Figure 3-1 for an evaluation of Matrix Center protocols for the treatment of MA abuse and dependence.

Figure 3-1

Evaluating the Matrix Model

The Center for Substance Abuse Treatment has recently solicited applications to replicate and evaluate the Matrix 8- and 16-week protocols for the treatment of MA use disorders. This project will represent the first multisite evaluation of a specific psychosocial approach for the treatment of MA disorders. The goal of the project is to collect data on the clinical efficacy of the treatment approach, as well as cost effectiveness information on the two treatment protocols. This project is scheduled to be completed by September 2001.

Behavioral Family/Couples Therapy

People with substance use disorders often have extensive marital, relationship, and family problems. Stable marital and family adjustment is associated with better treatment outcomes. Inclusion of family members in treatment is based on the view that they can provide important support for the client's efforts to change and provide additional information about the client's substance use and other behavior. Interventions directed at improving marital and family adjustment have therefore been judged to have the potential to improve treatment outcome. Studies with alcoholics have supported this hypothesis, at least in part. Few studies have been attempted with stimulant users, however.

One randomized trial conducted with a heterogeneous group of substance users, many of whom were cocaine users, supported marital/family therapy as a means to improve treatment outcome (Fals-Stewart et al., 1996). Subjects were male substance users under current criminal justice supervision, who were living with a spouse during the past year, and who expressed a commitment to sustained substance abstinence. These individuals were randomly assigned to two treatment groups that received an equal number of therapy sessions across 24 weeks of treatment. For one group, those sessions focused exclusively on coping skills. For the other group, sessions consisted of coping-skills training plus behavioral marital therapy. The group that received marital therapy had better relationship outcomes (in terms of more positive dyadic adjustment and less time separated) than did the comparison group, and reported fewer days of substance use, longer periods of abstinence, fewer substance-related arrests, and fewer hospitalizations during the year after treatment. As might be expected, some of those differences dissipated over the course of the followup period, but this study illustrates an important role for behavioral marital therapy for stimulant users who have a relatively stable romantic relationship and who express a commitment to substance abstinence at the initiation of treatment.

Other Interventions With Supportive Research

Some additional interventions merit mention. Permitting women entering residential treatment to be accompanied by some or all of their children appears to improve retention. In a published controlled study on this topic (Hughes et al., 1995), women entering residential treatment for cocaine use who were permitted to have one or two of their children reside with them were retained significantly longer than women whose children were placed with the best available caretaker (300.4 vs. 101.9 mean days of retention). No other measures of outcome were reported.

Another study described procedures for improving treatment participation (Hall et al., 1994). Clients were cocaine-dependent male veterans. All clients began treatment as inpatients, typically for 2 weeks, and were then encouraged to continue therapy in the outpatient center of the same medical complex. Therapy consisted of individual and group therapy sessions. Participation in the outpatient regimen began either during the inpatient stay, in which case clients kept the same individual and group therapists throughout the inpatient and outpatient phases, or it began after the inpatient stay and subjects were assigned new individual and group therapists on entering the outpatient phase. Having participation in outpatient care begin during the inpatient stay resulted in somewhat better participation after hospital discharge, and significantly better initial (3 weeks) but not later cocaine abstinence.

Woody and colleagues reported that supportive–expressive psychotherapy may help the subset of clients interested in receiving such therapy to reduce their cocaine use (Woody et al., 1995). They studied a subset of newly admitted methadone clients who indicated an interest in receiving psychotherapy and were compliant with attending counseling sessions (less than half the clients admitted). These individuals were randomized to receive supportive–expressive psychotherapy plus substance use counseling or only substance use counseling. Supportive–expressive psychotherapy focused on exploring the role that substances played in relationship problems, troubling feelings, and other problems. Those who received psychotherapy used significantly less cocaine during the 24-week study than those who received only substance use counseling.

Finally, an intervention called "node-link mapping" may be helpful in reducing cocaine abuse (Czuchry et al., 1995; Dansereau et al., 1995; Joe et al., 1994). This intervention uses flowcharts and other methods to diagram relationships between clients' thoughts, actions, feelings, and substance use. Clients were individuals enrolled in methadone treatment who were randomized to receive standard counseling or node-link enhanced counseling. Those who received the node-link mapping appeared to reduce their cocaine use more during 6 months of treatment than those who received standard care, but the effect was not compelling. The node-link-mapping group was using more cocaine at the start of treatment. Although the node-link-mapping group showed a greater reduction from the start to the end of treatment than did the standard group, the absolute amount of cocaine use at the end of treatment was not significantly different. Further studies are needed in which these results are replicated in groups that start treatment with the same level of cocaine use or finish treatment with the node-link-mapping group using significantly less cocaine.

Other Models of Psychosocial Treatment

A number of other psychosocial models and approaches have been described, and some used quite widely, for the treatment of stimulant use disorders.

Network Therapy

Network therapy is based on the rationale that people can recover from substance use disorders if they have a stable social network to support them in psychotherapeutic treatment. In this model, clients receiving individual psychotherapy develop a network of stable, nonsubstance-using support persons, such as family, partners, and close friends. These support persons learn strategies from the therapist to support the therapeutic process for the individual being treated. They may interact regularly with the therapist, participate in treatment sessions with the client, and be involved in setting up treatment plans for the client.

Empirical evidence for network psychotherapy is scarce. Controlled trials of network therapy for cocaine or other substance use have not yet been published.

Acupuncture

Acupuncture is an ancient Chinese therapy in which thin needles are inserted subcutaneously at various points on the body. The technique is based on the belief that the body's normal functioning depends on a balance of two opposite polar energies that flow along lines of the body called meridians. Approximately 1,000 acupuncture points are aligned along these meridians, and their stimulation by the thin needles is believed to correct energy imbalances and enhance the body's natural capacity to heal itself. No controlled outcome studies have been reported supporting the efficacy of acupuncture for the treatment of cocaine or other stimulant use disorders (TIP 10, *Assessment and Treatment of Cocaine-Abusing Methadone-Maintained Patients* [CSAT, 1994b]).

Inpatient Treatment

Inpatient treatment has traditionally consisted of a 28-day stay in a hospital or residential treatment facility, during which daily activities such as self-help groups, group psychotherapy, and relaxation techniques were provided in a structured format. Generally supportive and sometimes confrontational in nature, inpatient treatment was aimed at combating clients' denial and initiating participation in the 12 steps of recovery originally delineated by Alcoholics Anonymous (AA). The major goals of most inpatient treatment programs are detoxification from the influence of chronic substance use and beginning the process of engaging with self-help programs such as AA and Narcotics Anonymous (NA). Treatment components include didactic learning about the processes of addiction and recovery as well as experiential techniques. Often the client's family is involved in special "Family Days" to acquaint them with these issues.

Originally developed for the treatment of alcoholism, the 28-day standard hospital treatment regimen was used especially in the early 1980s, when the numbers of clients seeking treatment for cocaine use disorders began to rise dramatically. This trend peaked in the mid-1980s, when more than half of clients in many private programs were being treated for cocaine abuse and dependence (Rawson, 1986). Most of these inpatient programs were adapted to treat cocaine users with few or no modifications from the alcohol regimens. In the mid-1980s, when cocaine use among middle-class Americans reached epidemic proportions, the standard 28-day inpatient treatment program was the most widely used treatment modality for this population (Rawson et al., 1991a).

Several hospital/residential treatment organizations did attempt to evaluate the effectiveness of their treatment programming for cocaine users. For example, Sierra Tucson, in Tucson, Arizona, conducted a program of outcome research during the 1980s designed to evaluate and improve the efficacy of its treatment efforts for cocaine users. The Hazelden treatment organization compiled an extensive database on the effectiveness of its treatment services with cocaine and other substance users. The Carrier organization has published a series of studies designed to evaluate the effectiveness of their treatment programs (Pettinati, 1991). Although the evaluations were not randomized clinical trials, the information collected in the reports supported the value of the treatment services.

The traditional 28-day inpatient treatment regimen was developed with little input from empirically based research. In the past several years, the use of such inpatient programs has been called into question by insurance providers, and subsequently their use has been steadily declining. As insurance coverage for inpatient treatment likewise began to dwindle, these programs became variable in length. Many programs closed, and others were forced to scale back on the services they provided. Currently, in many cases clients are covered for brief inpatient stays (up to 7 days) for detoxification purposes only, and psychosocial services have been limited. Inpatient treatment programs are widely variable in the credentialing of their staff, but nearly all employ some staff members who are themselves in recovery.

Long-Term Residential Treatment

Long-term residential treatment is used for substance users who are deemed to be in need of a structured support system for a sustained period. The structure provided by long-term residential treatment is designed to allow positive changes and stabilization in the client's attitudes and lifestyle. The durations of residential treatment programs vary; at one time, most programs were at least 1 year in duration, but today most are about 6 months, or even only 90 days. Most residential programs, both long-term and halfway houses, are staffed at least in part by people who are themselves in recovery.

Therapeutic communities (TCs), the most common type of long-term residential treatment, are residential treatment programs that usually use group activities directed toward effecting significant changes in the residents' lifestyles, attitudes, and values. They emphasize prosocial behavior and the assumption of responsibility for one's actions. Many referrals to TCs take place through the court system. In fact, TCs originally were designed for heroin-addicted clients with deprived socioeconomic backgrounds and long-term histories of criminal involvement.

Halfway houses are residential treatment programs providing transitional support for individuals who are usually progressing from a more restrictive environment, such as a TC, but who are not yet ready to function independently in the community. These individuals may not need the intensive structured environment of a TC but may not yet be ready for independent living. Requirements of halfway-house programs usually include specified community involvement, such as employment or enrollment in school, and abstinence from mood-altering substances. Evening group activities are structured around residents' work schedules.

Although relatively little empirical evidence exists supporting the efficacy of long-term residential treatment for stimulant use disorders, there is at least some reason to believe that it can be effective (Gerstein et al., 1994; Mueller and Wyman, 1997). Although clinical experience suggests that TCs are effective with a subset of cocaine users, to the Consensus Panel's knowledge no controlled clinical trials have been published supporting their efficacy in the treatment of cocaine-dependent individuals.

Pharmacological Treatments for Stimulant Abuse and Dependence

There is not yet an effective pharmacotherapy for cocaine use disorders, but this topic is being researched intensively. Because of differences in the neurochemistry of cocaine and MA, there is sound reason to believe that different pharmacotherapies may be needed to treat those two forms of stimulant use (Ling and Shoptaw, 1997). However, because both drugs produce similar effects on the brain's dopamine levels, promising medications for the treatment of cocaine use disorders are also being examined for the treatment of methamphetamine use.

Clinical research on pharmacotherapies for MA use disorders is just getting under way. Medications are being sought to address a range of indications. There is interest in developing agents that can alleviate the medical/psychiatric symptoms caused by MA intoxication and withdrawal. For example, antidepressant medications have been found useful in the treatment of individuals who have discontinued their use of MA (NIDA, 1998c). Also, there is interest in developing medications to treat MA abuse and dependence. Ongoing trials are currently assessing dopaminergic (i.e., dopamine-mediated), serotonergic (i.e., serotonin-mediated), and other compounds (CSAT, 1997).

Pharmacotherapy research for cocaine use disorders was spurred initially by an open-label trial followed by a double-blind, randomized trial supporting the efficacy of desipramine, a tricyclic antidepressant, in producing short-term reductions in cocaine use and craving in outpatients (Gawin and Kleber, 1984; Gawin et al., 1989). In the randomized trial, 59 percent of cocaine-dependent clients treated for 6 weeks with desipramine achieved 3 or more weeks of continuous cocaine abstinence compared with 25 percent of those treated with lithium and 17 percent of those who received placebo (Gawin et al., 1989). Unfortunately, those promising results have not been replicated in subsequent controlled trials with desipramine (e.g., Carroll et al., 1994a; Weddington et al., 1991) or imipramine (Janimine), another tricyclic antidepressant (Nunes et al., 1995). Evidence that clients with less severe cocaine dependence may benefit from treatment with desipramine and imipramine was presented in at least two reports and merits further study (Carroll et al., 1994a; Nunes et al., 1995).

Other antidepressants that have been investigated in primary cocaine users include fluoxetine (Prozac) (Grabowski et al., 1995), maprotiline (Ludiomil) (Brotman et al., 1988), and gepirone (Jenkins et al., 1992). Studies are still in progress with some of these compounds, but none has demonstrated reliable efficacy in reducing cocaine craving or use in controlled trials. Because of cocaine's very pronounced effects in the dopamine system, a variety of different dopaminergic compounds has been investigated, including amantadine, bromocriptine, bupropion, flupenthixol, carbidopa-L-dopa, mazindol, methylphenidate, and tyrosine (see reviews by Gorelick, 1994; Kleber, 1995; Mendelson and Mello, 1996). Open-trial data have sometimes looked promising, but no reliable positive effects have been observed with any of these compounds in randomized trials. The same is true for the anticonvulsant carbamazepine (Kranzler et al., 1995).

Buprenorphine is an opioid drug that is currently being evaluated as a treatment for opiate dependence in the same manner as methadone is used. In the course of this work, observations by several researchers suggested that buprenorphine might be an effective treatment for cocaine use disorders in the population that uses both opiates and cocaine (e.g., Kosten et al., 1992; Schottenfeld et al., 1993). However, other more rigorous clinical studies have failed to find that buprenorphine has efficacy in suppressing cocaine abuse (e.g., Johnson et al., 1995). Research continues on this topic. Currently, there is no convincing evidence showing that buprenorphine causes decreases in cocaine use or is associated with greater reductions in cocaine use than when methadone is used to treat clients who abuse opiates and cocaine (see Silverman et al., 1998).

Use of disulfiram therapy for clients who use both cocaine and alcohol looks promising. The majority of stimulant users meet medical criteria for alcohol dependence, and more than 90 percent are current alcohol users (Grant and Harford, 1990; Higgins et al., 1994a). Disulfiram therapy with social monitoring to ensure medication compliance was used as a standard component in the community-reinforcement-plus-vouchers treatment approach described above. A chart review was conducted on 16 cocaine-dependent individuals who received that treatment (Higgins et al., 1993a). Carroll and colleagues reported results consistent with these findings in a pilot randomized trial (Carroll et al., 1993b). In that study, disulfiram therapy was compared with naltrexone therapy in a population of 18 outpatients who abused cocaine and alcohol. Disulfiram therapy resulted in significantly greater reductions in drinking and cocaine use than naltrexone therapy. Finally, a larger randomized trial on the efficacy of disulfiram therapy was completed recently, and again cocaine use was significantly reduced by disulfiram therapy (Carroll, 1996). A detailed protocol for use of disulfiram therapy with cocaine users is provided in the NIDA manual on community reinforcement plus vouchers mentioned above (Budney and Higgins, 1998).

Finally, an exciting area of research currently being pursued in the basic-science laboratory using nonhuman subjects focuses on the development of potential vaccines against cocaine use disorders in the form of enzymes or catalytic antibodies. These novel approaches may hold greater promise than more conventional approaches (Ling and Shoptaw, 1997).

4 Practical Application of Treatment Strategies

The preceding chapter describes what is known about the effectiveness of treatments for stimulant use disorders. This chapter focuses on how to use that information to improve treatment efforts with stimulant users. Whenever possible, treatment recommendations will employ strategies with empirical support. However, because many stimulant use treatment issues have not been systematically researched, the recommendations of the TIP's Consensus Panel as augmented by field review feedback are the basis for other recommendations.

Individuals seeking help for stimulant dependence receive the majority of their treatment in outpatient treatment programs. Accordingly, the treatment strategies described emphasize techniques employed in outpatient treatment programs. However, many if not most of these strategies and techniques can be integrated into programs other than structured outpatient treatment programs.

This chapter describes the key components of the stimulant use disorders in chronological order as they typically unfold to provide clinicians delivering treatment with a clinical road map. Treatment recommendations are offered to systematically address these clinical issues as they emerge.

This chapter is written with the assumption that abstinence from all mood-altering psychoactive drugs is the ultimate treatment and program goal. Although there is debate about the universal appropriateness of this position, the current treatment system in the United States is founded on this philosophy. This chapter also assumes that structured outpatient treatment will be viewed as one interdependent component of a larger substance use disorder treatment process and system. Many stimulant-dependent individuals experience medical problems or emergencies, psychiatric problems or crises, or various social, legal, or employment problems. As a result, although this chapter focuses on outpatient treatment of stimulant abuse and dependence, it acknowledges the critical importance of institutions and processes such as hospitals, medical/psychiatric management, and case management.

This chapter assumes that the client or the potential client is medically and emotionally ready for entry into outpatient care. Chapter 5 describes the medical/psychiatric factors that should be considered to ensure the safe admission of the client into an appropriate care setting. In addition to these safety elements, there are other considerations for some stimulant user groups (e.g., pregnant women, women with children, adolescents, those with coexisting disorders, the severely impoverished, and homeless individuals) in which the use of outpatient strategies may not be appropriate. Treatment considerations for client groups with these special needs are discussed in Chapter 6. Client Worksheets referred to throughout this chapter are located in Appendix B.

Treatment-Seeking Stimulant Users

Stimulant use disorder treatment is a health care service, and stimulant users are the customers for that service. For a treatment program to effectively meet the needs of stimulant users, it is essential to understand the perspective of the "customer" as she approaches, enters, and participates in treatment. For example, opiate-addicted individuals frequently initiate contact with the treatment system when they have exhausted all resources and are in fear of the discomfort of opiate withdrawal. Their initial overriding priority is for "treatment" to provide medication to prevent or alleviate symptoms of withdrawal. Treatment programs incapable of responding to this client priority are unlikely to successfully engage opiate addicts in treatment.

Stimulant users approach the treatment system with a different set of priorities than opiate users, because for them avoidance of the withdrawal syndrome is not a major motivating factor. The priorities of stimulant users and the assistance they are seeking vary more greatly than those of opiate-addicted individuals. However, there are some common themes in the pretreatment perspective of many stimulant users.

"Bad Things Are Happening"

Admission interviews with stimulant users provide substantial evidence that one of the major treatment-seeking considerations for most individuals is that their use of cocaine or methamphetamine (MA) has resulted in negative consequences. These consequences include legal, job-related, medical, family/relationship, financial, and psychiatric problems. Frequently, the treatment-seeking stimulant user is focused more on receiving assistance with these problem areas than on achieving abstinence from cocaine or MA.

"Life Is Out of Control"

Treatment-seeking stimulant users frequently say, "My life is out of control." They point to their excessive behaviors associated with obtaining, using, and recovering from the use of cocaine or MA. These behaviors include, but are not limited to

- Extreme financial irresponsibility and/or initiation of illegal activities
- Lack of routine self-care behaviors (e.g., eating, sleeping, bathing)
- Excessive or personally aberrant sexual behavior
- Severely deteriorated employment/educational performance
- Escalating irresponsible behavior to family and spouse (e.g., spending subsistence money on drugs, failure to care for children, marital infidelity)

Accompanying these behaviors is an array of emotional turmoil including but not limited to

- Extreme cycles of euphoria and depression
- Intense anxiety, fear, guilt, and shame over medical, fiscal, legal, and personal relationships
- Anergia (lack of energy) and anhedonia (inability to feel pleasure) during periods of abstinence
- Anger, paranoia, and irritability during both periods of use and periods of abstinence

Cognitive Impairment/Clinically Significant Paranoia

As documented in Chapter 5, the use of stimulants produces significant cognitive impairment (van Gorp et al., 1998) and frequently is accompanied by severe paranoia. Users have difficulty concentrating, impaired short-term memory, and a relatively short attention span. Their ability to recognize the interconnectedness of their stimulant use and the chaos occurring in their lives is poor, and the pervading sense of paranoia (especially for MA users) makes the development of a plan to remediate their problems very difficult. In short, it is often difficult for stimulant users to make sense of what is happening to them.

Ambivalence/Skepticism About Treatment

Many uninitiated clinicians are frequently frustrated and angered by what they perceive as a "lack of motivation" or the presence of "denial" in treatment-seeking stimulant users. Few stimulant users enter treatment with unconditional enthusiasm about the goals and methods of treatment. A significant number present for treatment exhibiting hostility, skepticism about the need for treatment, and opposition to fundamental elements of many treatment plans (e.g., cessation of alcohol and secondary substance use, participation in self-help programs). Although many stimulant users profess a strong desire to discontinue stimulant use, their resistance to initiating recommended treatment behaviors is often a source of clinician frustration. The recognition of this ambivalence as an integral part of the stimulant addiction syndrome, rather than as a frustrating impediment to working on "the real treatment issues," can help clinicians recognize the importance of effective techniques to motivate positive behavioral change.

Craving

The experience of craving a substance is a hallmark of almost all substance use disorders. However, for stimulant users, the experience of craving plays an important role in the maintenance of drug use. The basic research findings described in Chapter 2 have documented the existence of neurophysiological correlates of stimulant craving. Virtually all stimulant users have experienced craving but have little understanding of the biological underpinnings of this subjective experience. The power and volatility of this craving response can be exceptionally difficult for some stimulant users to resist (especially those who use the rapid delivery ingestion methods of smoking or injection). For many, it is virtually impossible to imagine how "counseling" or some other form of nonresidential treatment will help with this "irresistible force."

This combination of behaviors, attitudes, and emotions is frequently present to varying degrees with the majority of treatment-seeking cocaine and MA users. Other issues that frequently are priorities to treatment-seeking stimulant users include the dysphoria that occurs upon discontinuation of stimulants, often referred to as "the crash"(Gawin and Kleber, 1986); the compulsive sexual behavior of many men (especially MA users), which is often reported as equally or more difficult to control than the drug use (Rawson et al., 1998b); and the discouragement about previous attempts in and outside of treatment to discontinue stimulant use, only to experience relapse to even more severe levels of drug use. This set of attributes is, in many ways, the "raw material" that programs need to address in the treatment of stimulant users.

Treatment Needs of Cocaine Users Versus Methamphetamine Users

In one site in Southern California, a group of 500 MA users and a group of 224 cocaine users were treated using the same outpatient protocol (the Matrix manual), in the same office, with the same staff during the same time period (Rawson et al., 1996; Huber et al., 1997). Although there were some substantial demographic and drug use differences between the two groups, their response to the outpatient treatment protocol was virtually identical. MA users presented with higher ratings of depression, hallucinations, and several other symptoms, and exhibited a more prolonged period of symptom remission. Yet the data collected during treatment and at followup suggested comparable response to this outpatient experience.

There appears to be little empirical rationale for designating any one of the following psychosocial approaches as being differentially effective for the two stimulant user groups. Therefore, the following treatment recommendations apply to users of both cocaine and MA.

Maximizing Treatment Engagement

Make Treatment Accessible

Programs should maximize treatment accessibility. Research has shown that placing treatment programs in areas convenient to clients is associated with lower attrition rates (Stark, 1992). Treatment should be provided during the hours and on the days that are convenient for clients. If the majority of clients do not work and find boredom and lack of daytime activities a significant contributing factor to substance use, daytime treatment programming may be helpful. For programs with a substantial number of working clients it is essential to have evening hours. Some programs may need multiple sets of hours for different client groups. Programs should be located in the areas that are accessible to clients, such as near public transportation and in a part of town viewed as safe for evening visits. In rural areas, small satellite sites may be needed to bring treatment closer to clients. Facilities should be accessible to individuals with disabilities.

Provide Support for Treatment Participation

Research has demonstrated the importance of addressing clients' concrete needs, including transportation, housing, and finances (Chafetz et al., 1970). Providers may find it necessary to establish protocols for rapidly addressing transportation barriers, such as by providing bus tokens, bus and cab fare, and vans. Some logistical barriers can be overcome by onsite services, through agreements with subcontractors, or by referrals. These can include providing onsite child care services, referrals to temporary shelters, vouchers for lunches, targeted financial assistance, assistance with paperwork regarding insurance, or filing for disability.

Referrals should not be limited to providing clients with the name, address, and phone number of an agency. Rather, referrals should involve advocacy: networking with agencies and organizations, calling those contacts, and setting up the appointments or visits.

Respond Quickly and Positively to Initial Telephone Inquiries

Stimulant users often make their first contact with the treatment system on the telephone or as a result of an exploratory visit to a counselor or treatment program. The manner in which the receptionist, intake worker, counselor, or other staff person handles the initial contact with the prospective client may decide whether or not the individual decides to enter treatment. Timeliness is an important factor too. Having a counselor, intake worker, or other staff person available to answer telephone inquiries immediately for as many hours per day as possible will increase admission rates. Telephone inquiries should be answered without substantial delay (stimulant users are often impatient individuals, who hang up when placed on hold). Taking messages and calling back later will frequently result in a failure to find the individuals; or, when contacted, they may have changed their minds. Having 24-hour hotlines can be useful because some stimulant users or their family members make their initial treatment inquiries during late night and weekend hours.

Seeking help at an addiction treatment program can be a profoundly difficult and painful act. In fact, in many cases, a family member or friend often makes the initial contact with the treatment program. Data from telephone initial contacts to the Matrix Center clinics in Southern California indicate that approximately 45 percent of all initial treatment inquiries are not made by the potential client, but rather are made by a family member or friend.

In some treatment programs, there is a belief that unless the potential client makes the call for the initial appointment, it is inappropriate to schedule one. This policy is apparently based on the belief that requiring substance users themselves to make the initial call helps to reduce client "denial" and decreases the "no show" rate. Analysis of the data from the Matrix calls indicated no significant difference in "no shows" depending on who made the initial appointment. Because ambivalence about treatment is common among treatment-seeking stimulant users, methods to "screen out" those who are "in denial" are counterproductive and impede treatment entry.

Schedule Initial Appointments With Minimal Delay

The individual's decision to seek help may last for only a brief period of time. As a result, many individuals seeking help fail to show up for their initial appointment if it is scheduled too far in the future. For these reasons, the interview should be scheduled as soon as possible and within 24 hours after clients initially contact the program (Higgins and Wong, 1998). Figure 4-1 discusses the importance of scheduling.

Programs may not always have the resources to conduct thorough intake interviews whenever contacted. However, programs can provide interim services or minimal contact. For example, a brief interview or a partial intake within 24 hours would be preferable to making an appointment several days after the initial contact. The interview might identify any acute needs that require immediate attention. Also, treatment programs can provide orientation meetings in lieu of waiting lists. If a waiting list cannot be avoided, staff members can telephone the individual to express concern for the individual's well-being, conduct mini-assessments, and provide basic recommendations, such as attending a 12-Step meeting. Such efforts can serve as a temporary bridge between the initial contact and a thorough interview and assessment. These interim services can take advantage of fleeting motivations for change.

Assessment Procedures To Enhance Treatment Engagement

Keep Assessments Brief

Many programs conduct multiple assessments, often by several members of multidisciplinary teams. This technique may be useful for some clients, such as those with complex coexisting disorders. However, stimulant-dependent individuals are often irritated by lengthy and repetitive assessments. For such clients, it is essential that client assessments conducted early in treatment not become onerous or barriers to treatment. Accordingly, initial assessments should be brief, focused, and nonrepetitive. There are several clinical assessment questionnaires for stimulant users available in Washton and Rawson et al. (Washton, 1991; Rawson et al., 1991b).

Figure 4-1
Schedule Appointments Quickly

Making an appointment within 24 hours of initial phone contact significantly increases the likelihood of showing up for an initial appointment (Festinger et al., 1995, 1996; Stark, 1992; Stark et al., 1990). Such research suggests that an accelerated intake is a low-cost and effective method of reducing the high attrition rates commonly observed between the initial clinical contact and intake interview.

Identify Clients' Expectations

It is important to identify clients' expectations, as well as their fears, concerns, and anxieties. For example, clients with previous treatment experiences may have anxieties about treatment failure. Programs should specifically make efforts to discover clients' worries and identify those issues that can be dispelled through information and education about the program and the treatment process. An important task here is to help eliminate their fear of the unknown.

Provide Clear Orientations

Individuals need a thorough, clear, and realistic orientation about stimulant addiction treatment. Clients should acquire a good understanding about the treatment process, the rules of the treatment program, expectations about their participation, and what they can expect the program to do for them. They should understand the basic components of treatment, the amount of time that will be involved, and what will happen next. An orientation can help to dispel fears and anxieties and can help to correct misunderstandings. Research has shown that providing effective orientations has a positive effect on program retention (Stark, 1992). Parts of the orientation may need to be repeated, because cognitively impaired stimulant-dependent clients may forget what they have been told.

Offer Clients Options

Motivation research demonstrates strongly and consistently that people are most likely to engage in an action when they perceive that they have personally chosen to do so. In order to perceive that one has a choice, there must be alternatives from which one can choose (Miller, 1985). Research suggests that substance use disorder treatment is more effective when a client chooses it from among alternatives than when it is assigned as the only option (Kissin et al., 1971). The ability to choose also seems to reduce client resistance and dropout (Costello, 1975; Parker et al., 1979). Thus, it is important to provide clients with options and negotiate with them regarding the treatment approaches and strategies that are the most acceptable and promising.

Keep It Simple

Initial information and instructions should be simple and clear. Although clients with stimulant use disorders will vary, many will have cognitive problems that will limit their ability to follow long and complex instructions or explanations. As noted above, it is valuable to include clients in selecting their treatment plan. However, once the selection is made, it is important to be clear about the specific requirements of the treatment recommendation and the next step in the treatment process.

Involve Significant Others

Whenever possible, family and significant others who support the treatment goals should be involved in the treatment process, including the initial assessment and intake processes. Significant others can provide collateral information regarding the individual's addiction and can be evaluated regarding their potential for helping to promote the treatment goals or for hindering progress.

Significant others should be provided with information about the addictive process, the treatment program, assessment results, and the next steps for themselves as well as for the client. Individuals who walk away from a significant other's assessment process without interacting with program staff members are likely to feel neglected and ignored. Also, significant others can be given information about their role in the addiction process. They also should be provided with information about codependency and self-help for significant others of addicted persons, such as that provided by Al-Anon.

Staff Behaviors To Enhance Treatment Engagement

Treat Clients Respectfully

Research has shown that welcoming clients and treating them with respect are important factors in improving immediate and long-term retention (Chafetz et al., 1970). Individuals who contact treatment programs should be treated with courtesy, friendliness, respect, and warmth. The importance of professional demeanor and a respectful attitude toward clients applies to all staff members with whom they have contact, including both clinical and nonclinical staff members. Potential clients should not, for example, be put on hold for long periods of time.

Although it is true that some stimulant-dependent individuals can be difficult and provocative, these clients are often frightened, disoriented, and cognitively impaired. All program staff members should consider the courage that it takes to seek help for treatment and the shame and anxiety that most clients experience entering treatment. Staff members should provide individuals with positive feedback for asking for help and seeking treatment.

Convey Empathic Concern

A review of treatment research noted that evidence of high levels of therapist empathy is associated with positive treatment outcomes, and empathy was the predominant therapist characteristic associated with positive treatment outcomes (Landry, 1995). Counselors should be warm, friendly, engaging, empathic, straightforward, and nonjudgmental. Although many clients with stimulant use disorders respond poorly to confrontation and pressure, counselors should not hesitate to provide advice, especially behavioral prescriptions. Advice and recommendations should be provided in a caring and helpful way, not in a controlling or confrontational fashion. Counselors should make deliberate attempts to exert calming effects on clients and remain mindful of clients' potential for extreme impulsiveness and irritability.

When stimulant users are treated in a calm and respectful manner, violent reactions are very rare. However, authoritarian and confrontational behavior by the staff can substantially increase the potential for violence.

Do Not Fight Resistance

Aggressive confrontations with clients must be avoided. It is counterproductive to fight resistance to change or resistance to treatment. Rather, take steps that promote the therapeutic alliance. (See Miller and Rollnick, 1991, for an excellent description of these methods.) Confrontational strategies designed to break through the "denial process" are counterproductive and may be dangerous with stimulant users (Lieberman et al., 1973; Milmoe et al., 1967). Client readiness for treatment and motivation for change are not static conditions. Rather, these are dynamic processes that can be increased (or decreased) through counselor efforts. Counselors should cultivate the motivation and readiness of clients for change and growth (Miller, 1995).

The Treatment Plan

Few data support specific recommendations on the appropriate duration for outpatient treatment episodes. Similarly, there is little empirical evidence to guide the selection of session frequency, session duration, or session format (group vs. individual) of outpatient services for cocaine/MA users. However, it does appear accurate to view treatment as a set of procedures that address a series of clinical issues in a fairly predictable sequence. To organize treatment strategies, it can be helpful to view the treatment process as consisting of (1) a treatment initiation period, (2) an abstinence attainment period, (3) an abstinence maintenance phase, and (4) a long-term abstinence support plan.

The Treatment Framework

One important function for any treatment plan is to give clients a clear structure and framework for their treatment experience. This structure sets up specific expectations and provides clients with the benchmarks they need to plan their treatment participation and measure their treatment progress.

Treatment Episode Duration

There are no data to clearly establish the proper duration for a treatment episode. However, it is necessary to provide clients with a framework for their treatment experience. Many of the research studies and those with extensive clinical experience have used 12 weeks (Carroll, 1996); 16 to 24 weeks (Rawson, 1986; Washton, 1989); or 24 weeks (Wells et al., 1994). In general, it appears that a duration of 12 to 24 weeks, followed by some type of support group participation, is a commonly used framework.

Session Frequency

There are reports in the literature that describe treatment plans scheduled from one session per week (Carroll et al., 1995b, 1995c) up to five sessions per week (Washton and Stone-Washton, 1993). One study that reported a negative treatment finding (Kang et al., 1991) reported that once-per-week psychotherapy was not an effective treatment for cocaine users. In general, the majority of reports have used multiple sessions per week (2, 3, 4) for at least the first several months, with a reduction to fewer (1, 2, 3) through month 6.

Session Length

The session lengths reported in the literature range from 30 minutes to 6 hours. In general, sessions of 45 to 120 minutes in length are most common.

Format

There is tremendous variability regarding the optimal session format. Treatment strategies described in the literature include individual therapy sessions (Higgins et al., 1993a); a specified combination of individual and conjoint sessions (Meyers and Smith, 1995); and a collection of individual, group counseling, classroom didactic sessions, and conjoint session formats (Rawson et al., 1995). Other organizations employ primarily a group approach with individual and conjoint sessions on an ad hoc basis (Washton, in press). There is no research to support the value of one format or combination of formats over another. The most compelling factor in choosing a format may be practical considerations. Individual sessions are generally more flexible for scheduling; group sessions are typically less expensive to deliver. Figure 4-2 presents considerations regarding treatment duration and format.

The only certainty about the treatment framework is that it is critical to give the client clear, specific expectations of his treatment involvement. If the expectation is two individual sessions for 4 weeks followed by one individual session for 8 weeks, or three group sessions per week for 24 weeks, this should be agreed upon in writing by the counselor and client. Clients should have a written schedule of expected attendance they can keep and give to family members who may be involved in treatment.

It does not appear appropriate to deliver these services on an ad hoc or as needed basis. The structure and expectation of a prescribed treatment regimen has clinical value, independent of the contents of the treatment materials. Certainly there may be modifications in the treatment plan as treatment proceeds, based on clinical progress or other considerations; however, the initial contract needs to be specific and clear.

Figure 4-2
Treatment Duration, Frequency, and Format

- **Research has not yet demonstrated the optimal duration, frequency, and format of treatment for stimulant addiction (Higgins and Wong, 1998). Some research suggests that longer treatment durations of 6 or 12 months are associated with better outcomes for cocaine-dependent individuals (Carroll et al., 1993a; Higgins et al., 1993a; Wells et al., 1994), but the research is not consistent and has not evaluated MA treatment.**
- Experience suggests that the duration of the initiating treatment is a minimum of several weeks. Most stimulant-dependent clients require 2 to 4 weeks to establish an initial period of abstinence and to overcome certain cognitive impairments. It is common for programs to encourage frequent visits during the first 2 to 4 weeks of treatment followed by less frequent visits.
- For clients with stimulant use disorders during this phase, the frequency of visits or sessions seems to be more important than their length. For example, three or four weekly visits of approximately 30 minutes appear to be more beneficial than fewer weekly visits that last longer. There is no evidence that clinic visits lasting more than 90 minutes are more effective than shorter visits. The greater frequency of clinic visits can help to establish behavioral accountability, contain impulses, and create daily structure.
- In practice, the most common format for stimulant use disorder treatment is group rather than individual therapies. Experience suggests that stimulant-dependent clients are capable of full participation in group-oriented therapies, although their low tolerance for frustration may make lengthy group sessions onerous. However, clients who are still paranoid and distrustful of others may not be willing to participate in group therapy, but may be willing to participate in individual counseling as an initial strategy and bridge into group treatment.

Strategies for Initiating Treatment

During the first days and weeks of treatment, it is important to remember that although stimulant users do not have to contend with the uncomfortable withdrawal symptoms of the opiate or the alcohol dependent client, they often are experiencing a set of dysphoric symptoms. The initial period of stimulant abstinence is characterized by symptoms of depression, difficulty concentrating, poor memory, irritability, fatigue, craving for cocaine/MA, and paranoia (especially for MA users). The duration of these symptoms varies; however, in general, they typically last for 3 to 5 days for cocaine users and 10 to 15 days for MA users. The severity of these symptoms and the dysfunction they produce may be sufficient to warrant hospital/residential care in order to establish a period of abstinence (see Chapter 5).

Initial Treatment Goals

The first several weeks of treatment have some relatively simple and straightforward priorities. They are to

- Establish treatment attendance
- Discontinue use of psychoactive substances
- Finish assessment of clinical needs
- Remediate stimulant "withdrawal" symptoms
- Resolve any immediate crises

The following recommendations for this period can be integrated into a variety of treatment frameworks.

Establish treatment attendance

Initiating a routine of treatment attendance involves giving the client a clear expectation of when and where this attendance should occur, what is going to happen when she attends, positive reinforcement when attendance occurs on schedule, and reminders when treatment is missed. During the initial weeks, clients will be early, late, come in under the influence, and frequently be in crisis and confusion. This initial period is an opportunity to "shape" appropriate behavior by reinforcing proper attendance. Staff should remember that simply attending the sessions is a major indication of treatment engagement and should be enthusiastically reinforced. There will be ample time to give clients corrective feedback on being late or missing sessions.

Schedule frequent contacts

Stimulant-dependent clients appear to benefit from frequent clinic visits, even if the contacts are brief. During the first 2 to 3 weeks, such clients should be scheduled for multiple weekly visits, even if the visits are 30 minutes or shorter (Higgins and Wong, 1998).

Use positive incentives to reinforce treatment participation

One of the most powerful strategies to increase treatment involvement and establish treatment engagement is to use incentives and other tangible positive reinforcers to reward progress in treatment (Higgins and Budney, 1997). The specific reinforcers will differ among client populations. Some clients prefer vouchers for retail items or coupons for fast food; others appreciate clothes for themselves or their children or rebates for payments. Some programs hold brief ceremonies or present certificates. Rowan-Szal and colleagues demonstrated the effectiveness of incentives for attendance at counseling sessions and substance-free urinalyses through the use of "stars" on an awards board (Rowan-Szal et al., 1994).

A primary message that should be conveyed to clients with stimulant use disorders is that they should return to the program, no matter what. Even if they use stimulants or other substances, they should return to treatment. Clients should be given appointment reminder cards, flyers, and schedules, with the message

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that they are expected to return and that they will always be welcomed back.

Call no-shows

Programs should routinely telephone clients who fail to show up for scheduled clinic visits. Clinic staff members should encourage clients to come in for the clinic visit and inquire about any possible crises that may have prevented their participation. Personal letters can also be used as reminders.

Create a positive environment

Research has demonstrated that providing treatment in smaller groups in friendly, comfortable environments is associated with lower attrition rates (Stark, 1992). Programs should be prepared for client feedback related to not belonging and not feeling comfortable. Clients with stimulant use disorders often feel that they do not belong in treatment because they are not addicted, because they do not like the appearance of the program, or because they do not feel that they can relate to the other clients.

Rather than simply assuming that these beliefs represent defense mechanisms, programs should take steps to improve the comfort level of the treatment program and experience. For example, whenever possible, programs should take steps to help clients maximize their ability to identify with other clients and not feel alone. This can include establishing a "buddy system" in which a somewhat seasoned client or alumnus is provided with opportunities to dispel fears and concerns about the program and treatment process. If "buddies" are matched according to assumed similarities in background, the process can help clients to feel that they can relate to others in the program.

Discontinue use of psychoactive substances

Encourage abstinence immediately

After an initial assessment interview, it can be useful to ask clients to agree to a "temporary" trial period of abstinence. Counselors can end the first interview with a specific plan for abstinence, such as abstaining from substances of abuse at least until the next clinic visit. Some form of structured preparatory treatment that can act as a bridge to the regular treatment program can be useful for clients who are unwilling to make such a commitment (Obert et al., 1997). These can include a preparatory group therapy that involves motivational enhancement techniques (Miller and Rollnick, 1991). These groups can be brief but frequent, such as three to five times per week, and can include urine testing.

It is important to recognize that an individual may be at different stages of readiness for change (Prochaska et al., 1992) regarding different substances. For example, an individual may have made the decision to stop using stimulants but is still contemplating the decision to stop drinking alcohol. The individual's hesitancy to enter treatment may reflect ambivalence about alcohol, not stimulants. A motivational group may help to move him from the contemplation phase to the decision and action phases with regard to alcohol.

Establish daily schedule

Time planning and scheduling should be promoted as an important way to deter spending a lot of time alone or having big blocks of time without planned activities. Typically, the daily routine of stimulant-dependent individuals revolves around seeking, using, and recovering from the effects of stimulants. To break this pattern, clients can be taught to use basic daily schedules through which they can provide structure and accountability to their lives. Counselors can provide clients with simple daily schedules such as those illustrated in Client Worksheet 1, Daily Schedule and Planner (see Appendix B for client worksheets). Clients should be vigorously encouraged to schedule and plan each day, especially during this early phase of treatment. Clients should be encouraged to plan time for clinic visits, 12-Step meetings, meals, healthy social activities, exercise, recreation, and leisure time.

Initiate urinalysis schedule

Immediately upon entering the treatment program, clients should be placed on a mandatory, vigilant, and frequent urine testing schedule. This schedule should continue throughout the treatment process, although the frequency of testing can be tapered as treatment progresses. Urine samples should be taken every 3 or 4 days so as not to exceed the sensitivity limits of standard laboratory testing methods (see the Strategies for Initiating Abstinence Section below for more on urine testing).

Encourage 12-Step participation

Clients should be encouraged to attend a 12-Step program meeting as soon as possible. They should be provided with a schedule of meetings that are easily accessible to them. Participation in self-help groups should be strongly encouraged but not required. Some individuals who refuse self-help participation nevertheless succeed in treatment. Thus, although self-help participation has been shown to be associated with positive treatment outcomes (Landry, 1995) and will be a great resource for many clients, it is not a necessary condition for all clients to succeed.

Finish assessment of clinical needs

Assess psychiatric comorbidity

Many stimulant users, especially those who use MA, will enter treatment exhibiting symptoms of depression and psychosis. Clearly not all stimulant users have co-occurring depressive illness or a psychotic disorder. With most stimulant users these symptoms subside over several days (for cocaine users) or several weeks (for MA users). However, some stimulant users do have a co-occurring depression or thought disorder. During the initial 2 weeks it is important to assess the possible existence of these other psychiatric conditions and, if present, initiate appropriate treatment, including medication. Individuals who express suicidal ideation or planning should be taken very seriously and should be treated as any other potentially suicidal person.

Assess stimulant-associated compulsive sexual behaviors

Research demonstrates an association between stimulant use disorders and a variety of compulsive sexual behaviors (Rawson et al., 1998b). These behaviors include promiscuous sex, AIDS-risky behaviors, compulsive masturbation, compulsive pornographic viewing, and homosexual behavior for otherwise heterosexual individuals.

Stimulant-dependent clients can have tremendous concerns and anxieties about the compulsive sexual behaviors that they engage in while using stimulants. Such clients often assume that they are the only ones who have experienced such feelings and engaged in such behaviors. As a result, they may believe that they are perverted sexually or have sexual identity issues. These feelings can be barriers to treatment engagement and retention. Thus, programs can provide education to stimulant-dependent clients about the associations between stimulant abuse and compulsive sexual behavior.

Remediation of stimulant "withdrawal" symptoms

During the initial several weeks of treatment it is important to remind clients that proper sleep and nutrition are necessary to allow the neurobiology of the brain to "recover." Giving clients "permission" to sleep, eat, and gradually begin a program of exercise can help to establish some behaviors that will have long-term utility. These behaviors will also help clients begin to think more clearly and begin to feel some benefit from the initial efforts in treatment

Provide crisis resolution

Clients should leave early treatment sessions with an assurance that the program can provide or secure immediate attention to critical medical and psychiatric problems. Clients should understand that the program will help them to obtain rapid access to medical and psychiatric evaluation and treatment if they need it. Written lists of community and self-help resources are helpful resources. Programs should develop and always have accessible for distribution a variety of self-help and community resource materials to provide to their clients. These materials should include the name, address, telephone number, and descriptions of 12-Step meetings, other self-help resources, medical clinics, social service agencies, temporary housing and shelters, battered women's shelters, and children's resources.

Strategies for Initiating Abstinence

During the first several weeks of treatment, most individuals stop or at least reduce their use of stimulants. However, even if people have difficulty achieving total abstinence, the first several weeks can be considered successful if treatment engagement is established and some initial steps toward abstinence are made. After the initial treatment engagement of 1 to 2 weeks, a clear focus is on the achievement of abstinence. Although there is no clear delineation between clients who are *initiating* abstinence and those *maintaining* abstinence, the initiating period occurs roughly from 2 to 6 weeks into treatment.

The primary goals of strategies used in this phase of treatment are to (1) break the cycle of compulsive, repetitive stimulant use, (2) initiate a period of abstinence from all substances of abuse, (3) encourage the establishment of behaviors that support abstinence, and (4) initiate changes in attitude, behavior, and lifestyle that help maintain abstinence. The following section describes techniques for accomplishing these goals.

Establish Structure and Support

Initiating abstinence from stimulant addiction is not a mental exercise but a specific plan of behavioral action. To initiate this plan, a basic structure and daily routine must replace the lifestyle dominated by drug seeking, using, and recuperating. Structure, stability, and predictability are provided by a simple plan that clients can follow on a daily basis. The daily structure should incorporate and build around the client's participation in the treatment program. This will include establishing short-term goals, frequent counseling sessions, frequent urine testing, developing a support system, and time planning (Washton, 1989).

Short-term goals should be set immediately and should be reasonably achievable. One such goal is complete abstinence from all substances for 1 week. Because many stimulant-dependent clients engage in binge use, a comparable goal is to achieve a period of abstinence approximately twice as long as the usual time period between binges. Brief, frequent counseling sessions can reinforce the short-term goal of immediate abstinence and establish a therapeutic alliance between the client and counselor. Events of the past 24 hours are reviewed in each session, and recommendations are provided for navigating the next 24 hours. Establishing a social support system and conducting frequent and regular urine testing are also critical to providing structure, support, and accountability.

Daily schedule

The daily scheduling exercise described in the previous section continues to be an extremely important organizing strategy through this phase of treatment. Proactively planning time is a direct counterpoint to the impulsive, free-form lifestyle of the substance user. Clients should write down their schedules during session time, and session time should be used to review compliance with the schedule prepared in the previous session. Many clients will find this task difficult and may resist this "regimentation" of their time. However, if counselors reinforce successive efforts to follow such schedules, compliance will improve.

Conduct urine testing

Stimulant-dependent clients in outpatient programs need structure that provides support for engaging in healthy behaviors. Urine testing is part of that structure. It should not be presented or used primarily as an investigative tool or as a method to test the honesty of clients. Rather, it should be used and presented as a means of support for initiating and maintaining sobriety.

Urine testing should be conducted for the primary stimulant and for secondary substances. Testing should be conducted in concert with the clinic visits. During this phase of treatment, urine testing should be conducted no less than once a week. Tests should be spaced so that the results are obtained from a previous test before conducting the next test, which generally means spacing tests no more frequently than every 3 days. Testing should be randomly conducted, although it is advisable to test on days that closely follow periods of high risk, such as holidays, paydays, and weekends. To ensure that the urine is a valid sample from the client, testing should be either observed or monitored through the use of temperature strips.

Address Secondary Substance Use

Most stimulant-dependent clients also use some other substance, such as alcohol or marijuana. They often do not perceive their use of a secondary substance as problematic. Indeed, for many clients, their secondary substance use may not have been associated with adverse consequences or compulsive use. As a result, such clients need help to identify the connections between the use of other substances and their stimulant addiction. Clients should learn that using another substance increases the likelihood of relapse to stimulants (Rawson et al., 1986; Carroll et al., 1993a, 1993b).

Clients should learn that some secondary substances of choice, such as alcohol, can have a disinhibiting effect and lead rapidly to stimulant use (Higgins et al., 1996). A similar finding has anecdotally been reported by MA users with regard to their use of marijuana (Rawson et al., 1996). Clients should learn that the dose or the frequency of use of the secondary substance is not important, but that disinhibiting effects and potent conditioned responses and cues can occur at low doses. Achieving abstinence helps clients learn to develop substance-free coping mechanisms.

Clients can be helped to examine some of the reasons for why they use secondary substances. For example, some stimulant-dependent women use alcohol as a way to tolerate an abusive situation. Also, clients can be taught avoidance strategies for the secondary substance, such as eschewing high-risk situations where alcohol will be served.

Clients are sometimes ready for treatment for the primary substance of choice but not their secondary substance. Thus, secondary substance use is common during this phase of treatment. Although programs should promote abstinence from all psychoactive drugs, clients who use their secondary substance should not be discontinued from treatment solely because of this use. Rather, they should receive treatment strategies to help them decrease the likelihood of doing so in the future.

Establish Contingencies

Contingency management (described in Chapter 3) reinforces desired behavior by providing immediate consequences. It can be used to

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improve compliance with treatment components and to promote abstinence. It sets concrete goals and emphasizes positive behavior changes.

In contingency management, a specific target behavior, such as providing stimulant-free urine samples, is selected. The target behavior should be easily measured. Next, a specific and desirable contingency is identified and selected as a reward for each time that the target behavior is accomplished. The reward should not be exchangeable for cash, but can have a cash equivalent, such as a cash-equivalent voucher system or nonrefundable movie passes. The link between the targeted behavior and the reward should be specified. Finally, the agreement should be documented in a written contract and should specify the duration and any changes over time in contingencies. Contingency management interventions have been shown in controlled research studies to be effective for helping cocaine users to achieve and sustain abstinence (Higgins et al., 1994b; Silverman et al., 1996).

Initiate Avoidance Strategies

The process of identifying cues and triggers is dynamic and ongoing and will change over time. For example, as clients learn more about the associations between specific emotional states and stimulant cues, they can become increasingly sophisticated about identifying and avoiding or defusing potential triggers. However, there are several strategies that should be used very early in the treatment process to help clients to avoid certain external or environmental cues that are likely to be potent triggers for stimulant cravings and urges (Washton, 1989). These include discarding drugs, drug paraphernalia, and materials related to substance use; breaking contact with dealers and users; avoiding high-risk places; and developing basic refusal skills.

First, if the client has not already done so, a specific action plan must be developed to find and get rid of all substances (including alcohol) and drug-related paraphernalia. Clients should be encouraged to accomplish this task with the help of a family member, sober friend, or 12-Step sponsor to ensure that all drug-related items are found and permanently discarded. In addition to objects used to prepare or inject stimulants, materials associated with drug use that should be discarded include phone numbers of dealers and prostitutes, pornographic videotapes, containers used to hold drug supplies, mirrors or special tables used to cut stimulants, and weighing scales.

Second, it is essential for clients to develop specific action plans to break contacts with dealers and other stimulant users. When spouses and significant others are themselves stimulant users, it is important to develop a plan to assertively encourage the significant other to also seek help.

Third, an action plan should be developed to help the client avoid high-risk places. This involves identifying places strongly associated with stimulant use and making specific plans to avoid them. This may include taking different routes home from work, going to certain locations at times different than normal, or using a "buddy system" when going to a high-risk area. Finally, a plan of action should be developed to deal with confrontations with acquaintances who are still using stimulants. Clients should prepare specific drug-refusal statements that can be used when they encounter drug-using friends and practice with their counselor and fellow group members. This action plan must include immediately leaving the situation after the encounter. Client Worksheet 5, Action Plan for Avoidance Strategies (see Appendix B), can be used to assist clients develop strategies to avoid potent high-risk cues and triggers.

Provide Client Education

Clients with stimulant use disorders often do not understand many of the things that they have experienced as a result of their stimulant use, such as impulsive behaviors, anger and hostility, and cognitive deficits. They require education to help them understand the learning and conditioning factors associated with stimulant use. Similarly, they need information about the impact of stimulants and other substances on the brain and behavior, such as cognitive impairment and forgetfulness. Information about stimulant-induced behavior can help them understand the episodes of anger, hostility, and sexual compulsivity.

Clients, especially those with MA use disorders, should be educated about the early abstinence syndrome and protracted abstinence. Also, they should learn how their secondary substance of choice has an important role in relation to relapse to stimulant use. They require education about the biopsychosocial processes of addiction, treatment, and recovery. They should also learn about the stages of treatment and recovery, as well as the specific tasks, goals, and pitfalls of each.

Teach Basic Conditioning

Although many clients with stimulant use disorders in early treatment phases have poor retention of information and temporary cognitive deficits, they can understand basic information about cues and triggers. They can be taught how conditioning factors can elicit drug cravings and urges, that these cravings and urges are a natural part of early abstinence, and that there are methods to deal with them. Clients with stimulant use disorders should be provided with basic education about the conditioning process and how this process is applied to their disorder.

These educational efforts should describe basic conditioning factors related to stimulant use as described in Figure 4-3.

Identify Cues and Triggers

Stimulant (and secondary substance) use becomes strongly associated with certain people, places, objects, activities, behaviors, and feelings. Because clients with stimulant use disorders may have engaged in stimulant use hundreds or thousands of times, their daily life is filled with numerous reminders or cues that can trigger stimulant cravings and stimulant use. Although it is common for many clients to have some of the same cues and reminders, such as seeing the drug or the dealer, there are wide differences among clients regarding the specific type, strength, and number of cues. Accordingly, it is important for counselors to help clients to acknowledge and identify the cluster of cues unique to their lives.

The primary tasks here are to teach clients how cues are developed and how these cues can trigger drug craving and use, and to encourage them to actively identify their cues and triggers. This can be accomplished through exercises and worksheets. Client Worksheet 2, Identifying External Cues and Triggers, and Client Worksheet 3, Identifying Internal Triggers, can be the basis of exercises to help clients accomplish these tasks.

Develop Action Plan For Cues and Triggers

External and internal cues often pervade every aspect of stimulant users' lives. As a result, clients should develop action plans with specific behavioral and mental steps to prevent cues from becoming triggers. They should be taught to avoid, wherever possible, external cues that strongly remind them of stimulant use. They should be taught to leave situations that are making them think about stimulants or experience cravings. They should be taught specific techniques to stop drug thoughts from becoming drug cravings. Finally, they should be taught immediately achievable techniques

Figure 4-3

Basic Conditioning Factors in Stimulant Use

- **Stimulant cravings are the predictable results of chronic stimulant use and typically continue long after the stimulant use is stopped.**
- Stimulant cravings can be triggered by people, places, situations, things, and feelings that were previously associated with stimulant use. Anything that reminds clients of stimulant use can be a trigger for stimulant cravings.
- Stimulant cravings are typically strong during the early abstinence period and become less frequent and severe over time. They lose their power only when not reinforced by stimulant use.
- The strength of cravings does not diminish merely through the passage of time but because clients do not give into to the cravings when they occur.
- Complete abstinence from all psychoactive drugs is the best way to ensure the most rapid and complete extinction of stimulant cravings.
- Determination and willpower are poor defenses against cravings. Rather, specific actions must be taken to counteract cravings and urges whenever they occur.
- Cravings and urges are always temporary. They are usually fleeting sensations lasting no more than a few minutes and tend to disappear quickly when immediate action is taken to remove oneself from the situation that has prompted the craving.

Source: Adapted with permission from Washton, 1989, p. 107.

that can defuse stimulant cravings from leading to drug use. Client Worksheet 4, Action Plan for Cues and Triggers, can be a valuable part of such educational efforts.

Enlist Family Participation

Families and significant others should be encouraged to participate in treatment. The family should receive education about the addictive process, its role in the process, and its role in the treatment and recovery processes. Family members also need information about the effects of stimulants on the brain and behavior in order to understand the stimulant-induced behavior. They should receive a primer on the classical conditioning aspects of stimulant use disorders, and look at cravings as a conditioned response.

The information should be clear and simple, and not too conceptual or abstract. The ideal format is a group psychoeducational session, consisting of a brief didactic session, followed by a video and a group discussion. The process should help to elicit discussions and examples about how what they heard and saw applies to them. Also, family participation can be an opportunity to do an informal evaluation of the substance use disorders of other family members. Through this process, program staff members may be able to identify certain treatment needs, which may require treatment or referral.

For clients who are actively working on achieving abstinence and who have a stable marriage or relationship with someone who is not using stimulants, involving the spouse and client in couples or relationship counseling can be valuable. This strategy can help to improve communication skills and the relationship. Research has shown that marital and relationship counseling can have positive effects on substance treatment (Landry, 1995; Stanton and Shadish, 1997). If relationship counseling is considered, the significant other must not have problems with substance use (excepting nicotine), and the significant other must agree with the basic treatment goals of abstinence and be willing to engage in behaviors that support sobriety. Some research results related to behavioral relationship therapy are presented in Figure 4-4.

Establish Social Support Systems

Clients with stimulant use disorders, especially during the early phases of abstinence, seem to have low frustration tolerance and appear to be restless in group sessions. But as soon as clients are able to do so (generally within a few days), they should be introduced to a structured and therapeutic group process, such as a beginner's recovery group. These groups can provide a preexisting support network and a forum for openly talking about early abstinence problems. At the same time, participation in 12-Step meetings, such as Alcoholics Anonymous (AA), Narcotics Anonymous (NA), or Cocaine Anonymous (CA), should be strongly encouraged. Clients can be given the short-term goal of attending ninety 12-Step meetings in 90 days.

Also, clients can be encouraged to establish or reestablish relationships with nonsubstance-using friends and family and, perhaps, to establish a "buddy system" with a healthy family member, friend, or 12-Step sponsor to call during crises.

Address Stimulant Use–Associated Compulsive Sexual Behaviors

Some clients with stimulant use disorders develop significant stimulant-induced compulsive sexual behaviors. These can include compulsive masturbation, compulsive or impulsive sex with prostitutes, and compulsive pornographic viewing. For these clients, interventions can be conducted, the result of which is to decrease the likelihood of both the compulsive sexual behaviors and stimulant relapse.

A first step involves asking clients to agree to a temporary sex abstinence plan for 2 to 4 weeks. Next, clients should be made aware that sexual feelings, thoughts, and fantasies are conceptualized as very high-risk triggers that will be acted out if they are not talked out. For people who have this problem, even normal, routine sexual thoughts and contacts can quickly become major triggers.

Figure 4-4
Related Research: Behavioral Relationship Therapy

A review of research evidence regarding behavioral relationship therapy and substance use disorder treatment outcomes (Landry, 1995) noted that

- Behavioral relationship therapy can improve the quality of interpersonal relationships, promote rapid reductions in substance use, enhance maintenance of sobriety, enhance treatment outcomes, and decrease the probability of treatment dropout. Relationship therapy both during and following treatment improves treatment outcomes.
- Spouse involvement in treatment yields better results than treatment without spouse involvement. Unilateral treatment of the spouse with the person with the substance use disorder has been found to increase the client's motivation for treatment.

Similarly, a meta-analysis of controlled studies that compared family therapy with other therapy approaches to substance use disorder treatment (Stanton and Shadish, 1997) noted that

- Family therapy was more effective and had higher retention rates than individual counseling or therapy, peer group therapy, and family psychoeducation.

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Programs should provide a safe environment for such clients to talk about these issues, either within the context of a group session or individual counseling. Discussions should be held about safe and unsafe sexual behaviors in regard to relapse prevention. Specific and clear recommendations should include not having sex with anyone with whom the client has gotten high, and not pursuing sex with anonymous or unknown partners. Client fears should be addressed, such as the fear that sex without drugs will be boring or impossible.

Many of the avoidance strategies used with psychoactive substances can be employed for these clients in relation to sexual cues. For some clients, the sexual behavior has a higher reinforcing effect than the stimulant.

Clients will need reminders to stay away from people, places, and things related to sexual behaviors. These can include porno shops, certain streets with prostitutes, and video shops. Also, clients should be educated about reciprocal relapse, in which one compulsive behavior is inextricably involved with another, and therefore, engaging in the behaviors associated with one condition can cause one to act out behaviors associated with the other condition.

Respond to Early Slips

It is rare for clients to go from active, full-blown stimulant addiction to complete abstinence. Rather, most clients go through a phase during which there are days without substance use and occasional days with substance use. In fact, substance use during this early transition from abusive or dependent use to abstinence should not be considered relapse because there was not a genuine period of abstinence from which to relapse.

Thus, substance use during this period of transition should not be characterized as relapse but rather as difficulty in breaking the pattern of stimulant use. Also, clients should understand that substance use is normal during this difficult phase, despite their hard efforts to the contrary. Program staff members should understand that substance use during this phase is not a sign of poor motivation but reflects multiple processes, including cues and triggers and a not-yet-stable brain. Slips can also be thought of as a behavioral indicator of conflict and ambivalence about stopping. At the same time, counselors should clearly communicate that they are not giving clients permission to use. Rather, they are making efforts to keep the client engaged in treatment.

Early slips should be considered opportunities for adjusting the treatment plan and trying other strategies. They can be opportunities for gaining an appreciation of the strength of cravings and triggers, and learning new methods to handle them. They can be an opportunity to examine if the treatment plan is adequate and appropriate or to increase the frequency of contact with treatment and/or the support system, such as self-help meetings and contacts with the sponsor. Some recommendations for guiding group discussions of slips are listed in Figure 4-5.

Early slips should not be considered as tragic failures but rather as mistakes. When slips occur, counselors can make a verbal or behavioral contract with clients regarding short-term achievable goals. This can include such simple tasks as agreeing not to use psychoactive substances for the next 24 hours, to attend a specific number of clinic sessions over the next couple of days, and to bring a significant other to treatment the next day. This process can involve having the client identify areas that need to be addressed or enhanced. It may be important to take a closer look at cues and triggers and determine if anything has changed.

Figure 4-5
Responding to Slips in Group Sessions

- Ask the person to provide a detailed account of the sequence of feelings, events, and circumstances that led to the slip.
- Encourage group members to ask the person for further details, and to help him identify early warning signs and self-sabotaging behaviors.
- Encourage group members to state their concerns for the individual.
- Encourage group members to offer advice and recommendations about preventing further slips.
- Ask the person to discuss his thoughts and feelings about what has been said in the group and what he intends to do differently.

Source: Washton, 1990a.

Strategies To Maintain Abstinence

Many stimulant users can discontinue the use of cocaine or MA for periods of time without the assistance of treatment involvement. As previously mentioned, "withdrawal" is a less important consideration for stimulant users than it is for users of substances that produce a physically uncomfortable withdrawal, such as opiates, alcohol, and benzodiazepines. For stimulant users, the trick is not in stopping, but in staying off, or avoiding relapse. In the treatment of stimulant users, achieving abstinence is the "warm-up act"; sustaining abstinence is "the main event."

The dichotomy between strategies to achieve abstinence and strategies to maintain abstinence is somewhat artificial and arbitrary because many of the same principles apply and many of the same techniques are used over the course of treatment. However, there are some issues that appear to increase in importance over the 1- to 4-month period typically needed for learning how to maintain abstinence. These are discussed below.

Protracted Withdrawal Symptoms

Once stimulant use is discontinued and a client's sleeping and eating habits are normalized, the majority of symptoms described as the "crash" typically lessens. However, the resolution of the crash symptoms does not signal that the brain is back to normal. Clinical observations show that there are significant biological and psychological symptoms that continue to hamper the functioning of stimulant users 90 to 120 days after discontinuation of substance use. The symptoms described include a mild dysphoria, difficulty concentrating, anhedonia, lack of energy, short-term memory disturbance, and irritability.

The existence of these "protracted withdrawal" symptoms has been the subject of some debate. Recently, evidence from positron emission tomography (PET) scan research has provided tangible evidence in monkeys that MA use produces very significant changes in brain functioning that last for more than 6 months (Melega, 1997a). The brain areas involved and the neurochemical deficits observed in these PET scans are consistent with the clinical symptomatology of this "protracted withdrawal syndrome." Although there is still reason to be cautious about specifying the precise cause or time course of this syndrome, there does appear to be neurophysiological evidence to support the factual basis of this phenomenon.

Predictable Relapse Scenarios

There are a number of common patterns to the relapse episodes of stimulant users who are attempting to maintain abstinence (Havassy et al., 1993). These include

- **Alcohol/secondary substance use leading to stimulant relapse**. Several studies have reported on the relationship between alcohol use and cocaine relapse, and other reports have supported this same pattern with alcohol and marijuana for MA relapse (Rawson, 1986; Carroll et al., 1993a, 1993b).
- **Return to substance-using friends**. The Panel's clinical experience suggests that returning to substance-using friends is a primary reason for a stimulant user's relapse.
- **Sexual behavior associated with substance use**. Particularly for men, sexual behaviors especially associated with stimulant use (e.g., prostitutes, pornography) are an important element contributing to stimulant relapse (Rawson et al, 1998b).
- **Craving elicited by external and internal stimuli**. The powerful influence of Pavlovian conditioned cues on the production of craving has been reported by many stimulant users as a contributor to stimulant relapse (O'Brien et al., 1993).
- **Negative affective states**. Emotional states can be important antecedents to relapse (Havassy et al., 1993). Stimulant users find anger, depression, loneliness, frustration, and boredom quite difficult to manage, and these feelings can initiate a behavioral sequence that ends in stimulant use.

Need for New Activities

Many stimulant users have spent a good portion of the years leading up to treatment entry with their lives revolving around substance use. Frequently, during the initial 6 to 12 months of abstinence they have little idea what to do with their lives. In particular, they often have very poor social and recreational behavior repertoires. The creation of new, positively reinforcing activities and interests is an important part of this period of treatment.

Abstinence Maintenance Techniques

The strategies recommended for maintaining abstinence draw primarily from the behavioral and cognitive–behavioral models described in Chapter 3. One overall theme in the following materials is that newly abstinent stimulant users can be taught a set of information and skills that can help them avoid relapse. The following strategies have been found to help stimulant users maintain their abstinence.

Teach Functional Analysis Of Stimulant Use

The purpose of functional analysis is to teach clients how to understand their stimulant use so that they can engage in problem-solving solutions that will reduce the probability of future stimulant use. The core components of a functional analysis are (1) teaching clients to examine the types of circumstances, situations, thoughts, and feelings that increase the likelihood that they will use stimulants; (2) counseling clients to examine the positive, immediate, but short-term consequences of their stimulant use; and (3) encouraging clients to review the negative and often delayed consequences of their stimulant use. Client Worksheet 29, Components of a Functional

Analysis, gives clients an overview of these components.

Maintain Positive Reinforcement

Employing contingency management agreements can help sustain initial treatment gains. When contingency management is used, the behavioral contract must be based on objective criteria such as urinalysis results and attendance at group therapy sessions. All specifics must be clearly detailed in the written contract. Systematic and consistent implementation of the agreement is crucial: Reinforcement must be delivered promptly when the contract is satisfied and withheld when it is not. Frequent, positive reinforcement of success is critical.

Client Worksheet 28, Sample Behavioral Contract for Stimulant Abstinence, can be modified and used to help meet the treatment needs of specific clients. As this sample contract illustrates, contingency management can involve receiving "points," credits, money, or other benefits or incentives.

Relapse Prevention Techniques

Relapse prevention techniques teach clients to recognize high-risk situations for substance use, to implement coping strategies when confronted with high-risk events, and to apply strategies to prevent a full-blown relapse should an episode of substance use occur (Marlatt and Gordon, 1985). The techniques involve several cognitive–behavioral interventions that focus on skills training, cognitive reframing, and lifestyle modification.

Relapse prevention techniques fall into several categories:

- Psychoeducation about the relapse process and how to interrupt it
- Identification of high-risk situations and relapse warning signs
- Developing coping and stress management skills
- Enhancing self-efficacy in dealing with potential relapse situations
- Counteracting euphoric recall and the desire to test control over use
- Developing a balanced lifestyle that includes healthy leisure and recreation activities
- Responding safely to slips to avoid escalation into full-blown relapse
- Establishing behavioral accountability for slips and relapse via urine monitoring and/or Breathalyzer™ testing

As reviewed in the previous chapter, there is a substantial body of literature on the use of relapse prevention techniques with stimulant users. The manual developed by Kathleen Carroll provides an excellent set of relapse prevention exercises, which can be directly applied in treatment settings (Carroll, 1996). The Matrix manual (Rawson et al., 1991b) previously described has a section on conducting relapse prevention training in a group setting and supplies handouts and instructions for their use. Washton has published a set of relapse prevention materials that can be easily incorporated into treatment programming (Washton, 1990a, 1990b). Also, Figure 4-6 sets out basic precepts to be used in addressing relapse. The following treatment themes are critical to the relapse prevention–based treatment strategies.

Provide Psychoeducation About Relapse Prevention

One major element of a relapse prevention approach is the delivery of information to stimulant users about a variety of use-related topics. One frequently used format for delivering this information is in psychoeducation groups. These groups consist of a mixture of education, peer support, and recovery-oriented therapy. The group leader provides a brief discussion or shows a short videotape on a specific topic that is relevant to the group participants. The group members are

Figure 4-6

Addressing Relapse

An integral aspect of relapse prevention involves eliminating and correcting dangerous myths and misconceptions regarding the process of relapse and the appropriate treatment response to it. The Consensus Panel recommends that the following concepts be incorporated into educational efforts for clients, counselors, and nonclinical staff members.

- Relapse is not necessarily a sign of poor motivation. Although relapse can be a sign of extreme ambivalence or poor motivation to quit using stimulants, even the most highly motivated and sincere clients can relapse. Relapse is a sign that something is wrong with the client's recovery plan, not with the client.
- Relapse is not a sign of treatment failure. It is a temporary interruption in the client's abstinence. It means that the client's recovery plan is incomplete and is a signal that the client is doing something that he shouldn't do, or that the client should be doing something that he isn't.
- Relapse is predictable and avoidable; rarely is it unpredictable. It is preceded by warning signs that the counselor and client should be trained to identify. It is the endpoint of a progression of attitudes and behaviors. It is interruptible and preventable.
- Relapse is not a single event invariably involving drug use. Rather, relapse is a process, as is addiction, treatment, and recovery. It has a beginning, a midpoint, and an end. Returning to drug use is the endpoint, not the beginning of the process.
- Relapse does not erase positive recovery changes. Clients need not "start over" but should avoid further drug use, remain in treatment, resume the recovery process where last left off, and enhance the treatment plan to avoid future relapses. A temporary setback can provide invaluable information about weaknesses in the treatment plan and suggest ways to prevent it from recurring in the future.
- The absence of relapse does not guarantee successful recovery. Abstinence is an opportunity to recover but is not a guarantee of recovery. Many clients who experience relapse make tremendous strides in personal growth and maturity, although some clients with uninterrupted abstinence never experience substantial changes or achieve lasting growth. Abstinence is an important first step in the recovery process but is not the final goal.

Source: Adapted with permission from Washton, 1989.

encouraged to discuss the topic as it is personally relevant to them. Also, the group leader encourages group members to discuss the problems, challenges, and successes that they are currently experiencing.

The topics typically discussed in a psychoeducation group for clients with stimulant use disorders include

- Cravings and conditioning
- Protracted abstinence
- Stimulants and the brain
- Identification of high-risk situations
- Developing coping and stress management skills
- Enhancing self-efficacy in dealing with relapse-risky situations
- Counteracting euphoria and the desire to test control over use
- Developing a balanced lifestyle
- Responding safely to slips to avoid escalation
- Establishing behavioral accountability

Many of these are addressed in the sections below. Some recommendations for running a relapse prevention group are presented in Figure 4-7.

Address High-Risk Situations

During the previous phase of establishing abstinence, clients should have learned skills for negotiating high-risk situations. In particular, clients should be able to identify cues and triggers, develop action plans for cues and triggers, and deal with early abstinence symptoms.

Enhance Self-Efficacy Regarding High-Risk Situations

Once clients learn to identify, manage, and avoid high-risk situations, the counselor and client should try to determine if the client is confident in her ability to use those skills in the real world. It is important to evaluate and have clients engage in self-evaluation to determine if they are overconfident regarding their avoidance and refusal skills, and to determine if they actually have more skills than they imagine.

Figure 4-7

Recommendations for Running a Relapse Prevention Group

- A relapse prevention group is a forum for clients to create a program of recovery and relapse prevention. The group provides a setting for sharing information about relapse and relapse prevention and spotting signs of impending relapse. Clients heading toward relapse can be redirected, whereas those who are on a good course can be encouraged. The group setting allows for mutual client assistance within the guiding constraints of the group leader.
- A group can be led by a therapist group leader and a recovering coleader. Ideally, the group leader also sees group members for individual sessions. The group leader must be clearly, actively, and unquestionably in control of the group and is responsible for setting the time limits and ensuring that all group members have opportunities to speak. The coleader can answer questions about and be an example of long-term sobriety.
- The group meeting begins with an introduction of new members, who are asked to give a brief history of their drug use.
- Following introductions, the group leader gives a casual and didactic presentation on a specific topic for approximately 15 minutes and/or presents an equally brief video.
- Next, relapse and recovery are discussed among the group members for approximately 45 minutes.
- For the next 30 minutes, the group leader elicits from the group members any recent problems that they want to discuss. Quiet and uncommunicative members are encouraged to talk about how they are feeling.
- At the end of the group session, the group leader ties up loose ends and summarizes the discussion. Unresolved issues may be acknowledged, and discussions can be carried over to the next scheduled meeting. Clients who appear troubled, angry, or depressed, and those who mentioned cravings can be asked to remain. The group leader and coleader can encourage such individuals to speak with their therapist as soon as possible. All sessions should end with a confidentiality pledge and a commitment to attending the next group session.

Adapted, with permission. The Matrix Center, Inc. *The Matrix Intensive Outpatient Program Therapist Manual.* Los Angeles: Matrix Center, Inc., 1995.

Client Worksheet 11, Evaluating Your Self-Efficacy Regarding Relapse, can help clients to evaluate how they think they would handle certain high-risk situations that they cannot avoid. Similarly, Client Worksheet 12, Increasing Your Self-Efficacy, involves role-playing exercises designed to simulate real-world high-risk situations and to increase the client's self-efficacy.

Counteract Euphoric Recall And the Desire To Test Control

Two important risk factors for stimulant relapse are euphoric recall and the desire to test control over stimulant use. Euphoric recall is the act of remembering only the pleasures associated with stimulant use and not the adverse consequences. Euphoric recall is a potent relapse risk factor because it minimizes clients' perceptions of stimulants' danger, promoting an ambivalence about quitting. For these reasons, so-called "war stories" that include euphoric recall and selective memory are powerful relapse triggers and should be strongly discouraged in recovery groups. Client Worksheet 18, Selective Memory About Stimulant Use, can help clients to explore this issue.

After beginning to feel healthier, more in control of their lives, and free of some of their stimulant-related problems, some clients feel that they are ready to try a new approach to stimulant use. For example, some may feel that if they are "careful," they can use stimulants without losing control over their use. Others may feel that this is a good time to try using stimulants "one last time," just to see if they can do it without escalating into compulsive use and loss of control. Clients should be taught that urges to test their control over stimulant use are a powerful relapse warning sign. Client Worksheet 19, Fantasies About Controlled Use, can be part of psychoeducation efforts designed to recognize these fantasies as warning signs that need to be addressed. Also, Client Worksheet 20, Those Ugly Reminders, can help clients make lists of negative consequences of stimulant use, which can be reviewed when they experience cravings, fantasize about controlled use, or romanticize their experiences with stimulants.

Respond to Slips To Avoid Escalation

Stimulant slips and relapses are mistakes, not failures, and indicate a need to adjust the treatment plan. After a slip, a relapse-specific session should be scheduled as rapidly as possible. The counselor should reassure clients that he has not given up on them. Counselors and clients together review and analyze the events leading up to the slip and identify which warning signs were present. Clients should be encouraged to consider the events of the previous few weeks, such as changes at work, at school, in social networks, or in family situations. Similarly, they should closely examine events and issues that occurred in treatment, such as getting new counselors, moving from one phase of treatment to another, or events happening to another client.

Clients should be helped to identify specific steps that can be taken to avoid future relapses in the event that a similar set of circumstances recurs. Most importantly, slips and relapses should prompt revisions in the treatment plan. Such revisions may include increasing the number of self-help meetings, participating in individual counseling for a brief period of time, or obtaining a 12-Step sponsor. Also, clients should receive recommendations and guidance to handle the negative thoughts and feelings caused by slips. Client Worksheet 7, Permission to Relapse, is a useful client handout for this purpose.

Teach Drug Refusal and Coping Skills Training

Stimulant users in recovery are often surrounded by individuals who continue to use: dealers, neighbors, friends, or family members. The ability to refuse stimulants when offered requires a special type of assertiveness, hence the need for a special type of assertiveness training. Drug refusal training reminds clients that individuals offering them stimulants do not have the client's best interests in mind. Rather, clients are taught to think of such individuals (even if friends or family members) as "drug pushers" who must be discouraged. Clients are taught that their primary goal is to refuse offers of stimulants. They are taught that their secondary goals are to reinforce their commitments to not use and to feel good about themselves for doing it.

This approach emphasizes the following elements that should be incorporated into encounters with individuals offering stimulants or inviting the client into high-risk situations.

- The first thing that should be said to the person making an offer of stimulants is "No."
- The person making the offer should be clearly told not to make such offers now or in the future.
- The client should make good eye contact and adopt an expression and tone that clearly indicates the seriousness of the request.
- Offer an alternative and healthy activity if the client wants to be with the individual (such as taking the children for a walk or going to a workout).
- Change the subject to a new topic of conversation.

In this approach, the counselor guides the client through three scenarios involving specific individuals, specific times of the day, and specific situations. Based on these scenarios, the client and counselor engage in role-playing exercises so that the client can practice these behaviors. Furthermore, clients are encouraged to engage in additional role-playing exercises with significant others or other appropriate people.

Other Strategies Useful in Maintaining Abstinence

Provide Relationship Counseling

The overall goal of relationship counseling is to develop effective communication skills to help couples achieve and maintain abstinence, change their lifestyle, increase enjoyment in their relationship, and learn better ways to problem solve. Specific exercises for conducting these sessions can be found in the Community Reinforcement Approach Manual (Budney and Higgins, 1998).

Provide Social and Recreational Counseling

These treatment exercises are designed to increase participation in prosocial activities that may serve as alternatives to stimulant use. This includes helping clients to develop interests and participate in recreational and social activities that do not involve stimulant or other substance use. Potential activities can be evaluated by the counselor and client according to how interesting they are to the client, how costly, to what degree they involve others, how much time they require, how likely the client is to engage in them, and how much physical exertion they require. Potential coparticipants are identified. The next step involves the development of an action plan to identify the specific steps necessary to engage in the activities. These should be incorporated into the treatment plan. Examples of these exercises are included in several of the aforementioned manuals.

Provide Social Skills Training

The social skills training efforts are used to help clients learn and practice skills that will facilitate nonsubstance alternatives for socializing, recreating, and coping with stressful interpersonal situations. The goal is to help clients experience more positive reinforcing effects and fewer negative, adversive effects from social interactions. The training can be especially helpful for clients who have problems meeting nonsubstance-using peers or interacting with coworkers, and who feel uncomfortable in social settings.

Social skills training techniques have been developed for anger management, anxiety in social situations, initiating pleasant conversations, and assertiveness training (Alberti and Emmons, 1982; Chaney, 1989; Monti et al., 1995).

Provide Vocational Counseling

This counseling is focused on helping unemployed clients locate a job, and helping improve the employment situations of clients with unsatisfactory jobs or jobs that are high-risk for relapse.

Promote a Balanced Lifestyle

Treatment, recovery, and relapse prevention efforts should address biological, psychological, social, and spiritual areas of life. Clients should be taught the value of recreational and leisure activities and how to incorporate them into their recovery program. Many recreational activities can offer opportunities for clients to learn or practice social skills, such as cooperation, teamwork, healthy competition, and leadership.

Vigorous physical exercise helps clients feel good about themselves, decreases anxiety and depression, increases appetite, and often helps clients sleep better. Clients should be taught the value of regular aerobic exercise and how to incorporate it into their daily or weekly schedule. Clients should be provided with a variety of options for exercise, such as dancing, walking, biking, jogging, tennis, swimming, skating, aerobics, and weightlifting. Client Worksheet 23, Exercise and Recovery, and Client Worksheet 24, Examples of Exercise Activities, can help clients understand the value of exercise in their recovery, review potential types of exercises, and learn to incorporate exercise into their recovery program.

Many clients in treatment for substance use disorders have problems related to nutrition and diet. Stimulants decrease appetite, leading to decreases in the intake of calories and nutrition. Clients with stimulant use disorders eat insufficiently, and when they do eat, often eat impulsively and eat foods with negligible nutritional value. As a result, these clients should receive a formal nutritional assessment conducted by a nutritionist as well as guidance regarding eating a nutritionally balanced diet, discarding patterns of infrequent and impulsive eating, and learning to plan and schedule nutritionally appropriate meals. Client Worksheet 25, Nutritional Self-Assessment, can help clients evaluate their own unhealthy patterns of eating and need for structure regarding nutrition.

Monitoring Disulfiram For Alcohol/Cocaine

In the Community Reinforcement Approach, all clients who meet the diagnostic criteria for alcohol dependence or who report that alcohol use causes problems in their attempts to achieve abstinence from stimulant use are offered disulfiram therapy. A typical disulfiram dose is 250 mg/daily unless the client reports being able to consume alcohol at that dose without a reaction. In such situations, the dose is increased to 500 mg. Disulfiram ingestion is observed by clinical staff members when clients come for urinalysis monitoring. Take-home doses are provided for the other days. (See

Figure 4-8 for related research on disulfiram therapy.)

The Role of Self-Help Strategies

Self-help strategies can be valuable components of all phases of treatment. Self-help strategies, especially those that focus on substance use, are especially valuable as ancillary activities that support the treatment goals of maintaining abstinence. In general, self-help programs help clients to develop appropriate social skills, create healthy social networks, establish healthy intimate relationships, and engage in substance-free healthy activities. They also provide opportunities to learn socially appropriate mores and norms, how to receive and give advice, and how to mentor others.

The most frequently used and available self-help strategy is the 12-Step approach. It is the rare city that does not have many AA group meetings every day, and most larger cities have numerous CA and NA meetings. Clients should be provided with information regarding the 12-Step process, such as meeting format, the spiritual component, the basic content and meaning of the 12 Steps, the role of the 12-Step sponsor, and the role of anonymity.

Although the Consensus Panel recommends participation in a 12-Step group, providers should not require clients' participation. Rather, it is better to encourage and recommend 12-Step participation, especially because 12-Step programs are self-described as voluntary self-help programs of recovery. Similarly, family members of clients should be encouraged to participate in 12-Step programs designed for family members, such as Al-Anon. Such encouragement can be provided by having meetings on site. Both clients and family members should receive lists with the addresses and times of meetings, and provide transportation when necessary.

Also, self-help strategies other than the 12-Step programs can be valuable components of treatment. Some are specifically related to substance use, such as Rational Recovery, Save Our Selves, and Women in Sobriety. These may be particularly helpful for individuals who are reluctant to participate in the 12-Step programs. These include such activities as church-related groups, cancer survivor groups, and domestic violence groups. Some research findings on AA are shown in Figure 4-9.

The Role of Psychodynamic Therapy

Substance use counseling generally consists of therapeutic efforts that focus primarily on solving present-day problems that interfere with abstinence and recovery. Although there is variation, counseling generally focuses on current issues and involves advice, guidance, and encouragement. It is typically conducted in group formats.

In contrast, psychodynamic therapy, typically conducted in individual session formats, focuses on intrapsychic processes that impair effective coping and damage relations.

Figure 4-8
Related Research: Disulfiram Therapy

An uncontrolled study by Higgins et al. (1993a) noted that supervised disulfiram therapy was associated with significant decreases in alcohol and cocaine use among outpatients with cocaine-related disorders. A subsequent controlled trial by Carroll et al. (1993b) provided support that disulfiram therapy can reduce cocaine and alcohol use in outpatients who use both substances.

Figure 4-9

Related Research: Alcoholics Anonymous (AA)

A comprehensive review of the research on AA reveals several important findings:

- Research demonstrates a strong association between AA participation that occurs during or following professional treatment and improvements in drinking behaviors and abstinence.
- Research suggests a strong association between increased frequency in attending AA meetings and improvements in drinking behavior measures, such as abstinence and decreases in alcohol consumption.
- Research suggests a modest association between increased participation in and affiliation with AA (such as obtaining or becoming a sponsor) and improvements in drinking behavior measures, such as abstinence, decreased drinking, and decreased relapse.
- Research suggests modest associations between AA participation and improvements in several areas of psychosocial functioning.

Source: Landry, in press.

Psychodynamic therapies differ greatly, but when used in substance use disorder treatment, they often assume that substance use is at least in part a strategy to self-medicate problems or a coping mechanism to deal with such problems as trauma, victimization, and low self-esteem.

Views differ regarding the appropriateness of individual psychodynamic therapy for clients with stimulant use disorders. As a result, the Consensus Panel makes the following recommendations. First, clients should be thoroughly evaluated to determine their need for this type of treatment. Are the client's individual treatment needs elicited through the treatment planning process best met by individual psychodynamic therapy or by providing basic skills to maintain abstinence? Second, clients should be thoroughly evaluated with regard to their readiness for psychodynamic therapy. Because psychotherapy can stimulate feelings and thoughts that may provoke relapse triggers, clients should be evaluated regarding their readiness to handle such triggers. Do clients have the emotional stability, relapse prevention skills, and social supports to handle this therapy? Third, if individual psychotherapy is introduced, it should be consistent and coordinated with other treatment strategies, especially group counseling and self-help involvement. For example, psychotherapy oriented to the stages of recovery can be especially useful (Wallace, 1992).

Overall, the Consensus Panel suggests that not all clients with stimulant use disorders are appropriate for, need, or want individual psychotherapy to establish or maintain abstinence. When provided, there should be an explicit treatment need, the client should have the requisite skills, and the therapy should support abstinence.

Next Steps

Because treatment should be based on the individual client's unique needs, the length of treatment should not be dictated by the number of weeks in the program. In particular, termination of the abstinence maintenance phase of treatment should be based specifically on achieving the treatment goals documented in clients' treatment plans.

The end of the abstinence maintenance phase is a good opportunity to help clients review their treatment experiences. Counselors should engage in activities and exercises that help clients to critically examine their treatment successes, the areas where they experienced problems, and the ways in which they addressed these problems. Similarly, counselors should help clients to evaluate the strength of their current recovery program and identify areas where they need strengthening. Through this process, the counselor and client should develop a continuing-care treatment plan that identifies remaining treatment needs and strategies that will be used to meet those needs.

Termination of the abstinence maintenance phase of treatment should be a transition to a lower level of care, not a termination per se. Abrupt termination should be avoided. Rather, programs should have or develop strategies that allow and encourage clients to remain connected with the program. Furthermore, programs should develop strategies that specifically educate clients about the continuing care treatment services available to them and that actively encourage clients to utilize these services. Ways in which programs can help clients to remain in contact with the program include

- Continuing care or aftercare group meetings—which clients can attend weekly or more often as needed
- Individual counseling or psychotherapy—which can be made available to clients on an as needed basis
- Family therapy—made available to clients and their families, and to families without the client, such as during relapse episodes
- Clean and sober alternative activities—focusing on recreation, leisure, education, and social activities (e.g., dances, field trips, summer barbecues, picnics, holiday events, lectures on topics not necessarily related to treatment or recovery)
- Treatment program alumni meetings—such as quarterly meetings during which all program graduates are invited
- Treatment program alumni clubs—in which programs sponsor and encourage regional groups of alumni to have regular meetings and events (programs can provide speakers on motivational and educational issues)
- Peer mentoring programs—in which program alumni help newly arriving clients by sharing experiences, advice, and program expectations
- Surveys and newsletters mailed to program alumni as a way to collect posttreatment data, to encourage participation in alumni activities, and to encourage contact with program, especially during times of need

5 Medical Aspects of Stimulant Use Disorders

This chapter addresses the symptoms, complaints, and other medical sequelae commonly seen in persons using various forms of stimulants (e.g., cocaine, crack, amphetamines, methamphetamine [MA]) who appear at hospital emergency rooms (ERs) and other medical settings, or who need specialized medical care while participating in residential or outpatient substance use disorder programs. The purpose of the chapter is to assist medical personnel in recognizing and treating problems that may arise for stimulant users with acute or chronic intoxication or in various phases of withdrawal after protracted use of these drugs and differentiating these from similar presentations of other medical and psychiatric conditions. Another emphasis is the need for establishing and ensuring linkages between medical facilities and appropriate, comprehensive substance use disorder treatment/rehabilitation programs.

In a meta-analysis of 555 consecutive cocaine-related visits to hospital ERs in four cities between 1989 and 1992, Schrank concluded that catastrophic complications directly related to the use of this stimulant comprise only a small fraction of the sequelae (Schrank, 1993). Deaths were relatively rare, occurring in only four patients. In this sequence of cases, there were no reports of myocardial infarction, intracranial hemorrhage, ischemic stroke, infarcted bowel, or pulmonary barotrauma. The most common reasons for ER visits by cocaine users were cardiopulmonary symptoms (usually chest pains or palpitations); psychiatric complaints, ranging from altered mental states to suicidal ideation; and neurological problems, including seizures and delirium.

Most of the patients had multiple problems, many related to intravenous substance use. Rapid medical intervention was crucial for cocaine users with seizures, hyperthermia, potentially lethal arrhythmias, or toxic delirium. However, most patients responded well to simple evaluation, observation, and supportive care. Pharmacological intervention was required in less than one-fourth of the 555 cases. Schrank emphasized the importance of recognizing concurrent use of multiple substances and the presence of cocaine or other drugs in victims of traumatic injury and in obstetrical patients.

MA users are much less likely than individuals using cocaine to arrive at the ER with such acute medical problems as cerebrovascular accidents, acute cardiac ischemia and failure, hyperthermia, or seizures. The major presenting symptoms for MA users pertain primarily to altered mental status, including confusion, delusions, paranoid reactions, hallucinations, and suicidal ideation. The rapid development of tolerance to its physiological effects among chronic MA users may explain the relative infrequency of cardiac complications in this group (Heischobar and Miller, 1991).

Toxicity, Addiction, and Other Adverse Reactions

The precise clinical effects of cocaine and MA depend on a complex mixture of the pharmacological properties and purity of the drug used; the dose, frequency of use, and route of drug administration; the user's state of intoxication or withdrawal and previous experience with the drug; and other concomitant medical and psychiatric conditions, including simultaneous use of other substances as well as personality attributes and expectations regarding drug reactions. All of these factors not only mediate drug effects, but also influence the user's susceptibility to substance abuse or dependence (Ellinwood and Lee, 1989; Gold, 1997).

Route of Administration

The method by which stimulants are taken—the route of administration—determines the dosage and the rapidity and intensity of effects. Route of administration also affects the potential for adverse reactions and the likelihood of addiction. The principal routes used to administer cocaine and MA are oral ingestion, nasal insufflation (snorting), intravenous injection, and inhalation of smoke vapors (smoking/inhalation). These stimulants can also be taken vaginally, rectally, or sublingually.

In general, smoking and intravenous use rapidly evoke similarly intense responses, whereas oral ingestion and intranasal administration are slower delivery mechanisms, causing lower and more gradually rising blood levels and less intense subjective responses. Indeed, cocaine is seldom taken by mouth in this country because first-pass hepatic biotransformation metabolizes 70 to 80 percent of the dose and substantially diminishes the drug's effects (Gold and Miller, 1997). When crack cocaine is smoked, a highly concentrated dose is rapidly delivered to the brain. Several studies have reported a close correlation between subjects' plasma levels and the subjective effects from single doses given to relatively naive users who have not developed tolerance (Ellinwood and Lee, 1989; Gold and Miller, 1997; Volkow et al., 1997a). As the efficiency of the delivery system increases, so does the intensity of both the pleasurable and adverse effects. Figure 5-1 depicts these general variations in response times according to the different routes of administration for cocaine and MA.

To some extent, the dangerous consequences and addictive potential of stimulants also reflect the route of drug administration. Oral ingestion of MA is thought to protect the user from cardiotoxicity (Cook et al., 1993), and the lower and more sloped peak blood levels achieved by this route are also thought to be responsible for lower rates of addiction (Gold and Miller, 1997). Also, cocaine appears to be less addictive if doses remain small, peak plasma levels and the onset of drug effects are slow, and unpleasant withdrawal effects are absent or minimal. Oral ingestion and, to some extent, intranasal routes fulfill these criteria of slower, less hazardous consequences (Gold, 1997).

Intravenous use is more toxic than intranasal or oral routes, but inhalation is generally perceived as the quickest and, from the user's perspective, the most desirable delivery method because smoked crack cocaine and ice MA produce the highest peak blood levels and the most potent subjective impact without attendant hazards from syringe needle use (Cho, 1990; Cook, 1991; Gold, 1997). Other investigators report that smoked ice does not seem to produce the same intense "rush" as injection. There is some indication that in utero exposure alters cocaine reinforcement properties for adults or, at

Figure 5-1
Effects of Route of Administration for Cocaine and MA

Route of Administration	Form of Drug	Onset of Action for Cocaine and MA	Duration of "High"
Oral	Powder/pill	10 to 30 minutes	45 to 90 minutes for cocaine 3 to 5 hours for MA
Intranasal	Powder	3 to 5 minutes	10 to 20 minutes for cocaine
Intravenous	Solution	5 to 10 seconds	10 to 20 minutes for cocaine 4 to 6 hours for MA
Inhalation	Crack cocaine/ Ice (MA)	5 to 10 seconds	5 to 20 minutes for crack 8 to 24 hours for ice

Sources: Cook, 1991; Gold, 1997; Gold and Miller, 1997; Sowder and Beschner, 1993.

least, increases rates of cocaine self-administration in the laboratory (Gold and Miller, 1997).

Different routes of drug administration also produce different side effects. Intravenous users frequently develop illnesses associated with the preparation of drugs for use (i.e., mixing/making) and the use or sharing of unsterile needles, including HIV infection, hepatitis, tuberculosis, lung infections and pneumonia, bacterial or viral endocarditis, cellulitis, wound abscesses, sepsis, thrombosis, renal infarction, and thrombophlebitis (Sowder and Beschner, 1993; Gold, 1997).

Nasal insufflation is associated with sinusitis, loss of sense of smell, congestion, atrophy of nasal mucosa, nosebleeds, perforation or necrosis of the nasal septum, hoarseness, and problems with swallowing. Crack users complain of throat ailments and a productive cough with black sputum (Gold, 1997; Gold and Miller, 1997). Intravenous use of MA is associated with greater severity of medical and social problems compared with other routes of administration (Sowder and Beschner, 1993).

Figure 5-2 compares differences in use and consequent problems between intravenous and nonintravenous MA users. Apparently, MA users recognize these route-related effects and tend to vary the routes of administration because the drug causes irritation to nasal mucosa and lungs (Center for Substance Abuse Treatment [CSAT], 1997). Even prolonged use of amphetamine-containing diet pills has resulted in ischemic colitis and pulmonary edema (Sowder and Beschner, 1993).

Differences Between Cocaine and MA

The major differences between cocaine and MA pertain to the rapidity of responses and the duration of their effects. The sought-after effects of MA can persist for hours, whereas those from cocaine are over in minutes. This has important consequences for the choice of drug and the patterns of administration adopted by users. The plasma levels from smoked crack cocaine both peak and decline rapidly, whereas those from smoked MA also peak relatively rapidly but decline more slowly because metabolism takes longer. Regularly repeated use may be more common among cocaine users trying to sustain the drug's effects, whereas withdrawal is more protracted for MA users (Cook, 1991; Gold and Miller, 1997). Figure 5-3 shows some of the differences between cocaine and MA.

The plasma levels of cocaine/crack peak and decline rapidly, with a terminal half-life of about 56 to 60 minutes. MA plasma concentrations also peak rapidly but remain high for much longer (Cho, 1990; Cook, 1991). In normal subjects, the plasma half-life of cocaine ranges from 40 to 90 minutes (Rowbotham, 1993).

Because the biological half-life of cocaine is relatively short, repeated dosing is necessary to sustain an effect (Gold and Miller, 1997). By contrast, repeated dosing with MA, before metabolism and elimination are complete, can

Figure 5-2
Dose Frequency Escalation Patterns, Cocaine and Amphetamine

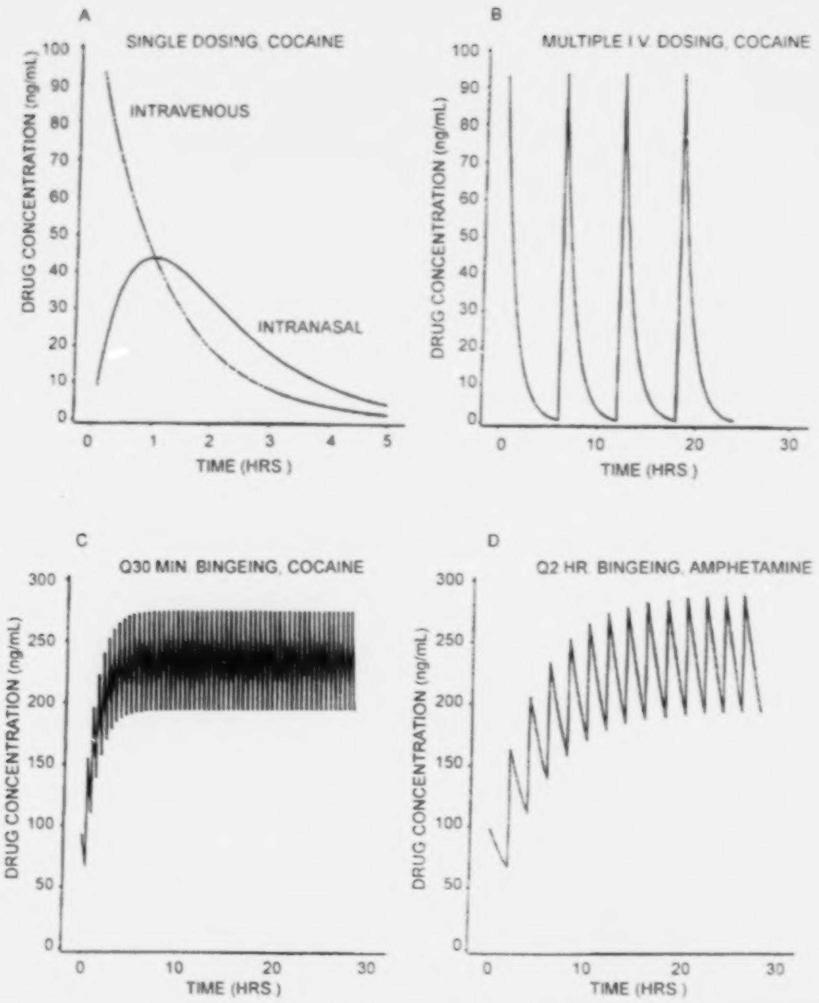

Figure 5-3

Differences Between Cocaine and MA

Cocaine	**MA**
Plant-derived	Man-made
Smoking produces a high that lasts 20 to 30 minutes	Smoking produces a high that lasts 8 to 24 hours
Is eliminated from the body in 1 hour	Is eliminated from the body in 12 hours
Used as a local anesthetic in some surgical procedures	Limited medical use

Source: NIDA, 1998a.

result in substantial accumulation of the drug in the body with increased likelihood for addiction (Cho, 1990; Cook, 1991).

Other factors in the growing preference for smokable forms of MA, as well as crack cocaine, include availability and price. Crack is generally less expensive and more available than powdered cocaine hydrochloride and produces, in the initial smoker, a very intense but brief rush described by some as a "full body orgasm" (Gold, 1997). Because ice costs less than other forms of MA per dose, and because the euphoria attained may persist for several hours, this form of MA delivers the most "bang for the buck." Because abuse liability increases as time before onset of action decreases, and the concentrations of the drug that reach the brain and receptor sites increase (Cornish and O'Brien, 1996), the current concern about increased use of stimulants pertains to both the smokable preparations (crack and ice) and to continuing intravenous use of both drugs.

Dose

The incidence and severity of MA- and cocaine-induced side effects and toxic reactions are also dose-related. As the dose increases, the profile of side effects progresses from mild excitement to more intense reactions, even psychosis (CSAT, 1997). Because tolerance develops rapidly to the desired euphoric effects, stimulant users nearly always escalate dose size and frequency of drug use in pursuit of the vanishing rush. If initial use was by oral or intranasal routes, users also tend to switch to intravenous administration or inhalation, methods that promise more rapid response rates and peak plasma levels (Ellinwood and Lee, 1989).

Chronic MA users may consume as much as 15 grams of the drug per day in doses exceeding 1 gram every 4 hours over a 24-hour period. Because the conventional dose is 10 mg, doses of 150 mg to 1 g would ordinarily be highly toxic to naive users (Cho, 1990). There is, however, considerable individual variation in toxicity and overdose from stimulants. Although general ranges have been established for lethal doses and blood levels, reactions are unpredictable (Gold and Miller, 1997).

The lethal dose of cocaine for 50 percent of novice users (LD_{50}) is 1.5 grams. The LD_{50} for MA has not specifically been established, and there is significant individual variability to its toxicity. For example, doses of 30 mg can produce severe reactions, yet doses of 400 to 500 mg are not necessarily fatal. Reported tissue levels of MA in fatalities, nonetheless, have ranged from 1μg/mL to over 14 μg/mL. Reported blood levels have also ranged from 27 μg/mL to only 0.6 μg/mL (Mori et al., 1992).

Purity of the Drug

The purity of the stimulant used also influences the rate and completeness of its absorption and effects. The purer the drug, the greater the effects. "Street" drugs, however, are seldom entirely pure. The purity of confiscated cocaine hydrochloride intended for oral or intranasal consumption generally ranges between 20 and 80 percent; the purity of intravenous cocaine preparations can vary between 7 and 100 percent; and for freebase or crack intended for smoking, from 40 to 100 percent (Gold and Miller, 1997). Most seized batches of MA have 40 to 70 percent purity (Burton, 1991; CSAT, 1997).

Adulterants are added to cocaine to increase its weight by cutting or substituting less expensive but similar-tasting and acting products that will maximize profits for the dealer while still satisfying the customer. In general, the adulterants in cocaine do not pose serious health-related problems, although these cannot be completely discounted (Schrank, 1993). Cocaine is most often cut with mannitol, lactose, quinine, glucose, or other inert compounds for weight, and with caffeine, lidocaine, other stimulants, anesthetics, or hallucinogens for taste and effect (Schrank, 1993; Gold, 1997).

The manufacturing processes for illicit MA and ice are often crude and can involve many impurities and contaminants that do pose serious health consequences. Until recently, most of the MA sold on the street was manufactured from phenyl-2-propanone (P2P), a method of synthesis in which lead acetate is used as a chemical reagent. The large quantities of lead in the final product can result in symptoms of hepatitis, nephritis, and encephalopathy (Allcott et al., 1987). Two outbreaks of lead poisoning in Oregon in 1977 and 1988 involving a total of 14 cases among intravenous MA users were blamed on the lead acetate used in the P2P manufacturing process. Testing revealed the presence of 60 percent lead by weight in one case (Irvine and Chin, 1991).

The typical clandestine manufacturing process for MA has changed over the last 12 years—from the P2P method—to an ephedrine-based method and, more recently, to pseudoephedrine and phenylpropanolamine processing (CSAT, 1997). The difference between these two primary synthesis methods is primarily the precursor chemicals used.

The newer and more popular ephedrine method, which accounted for 89 percent of production in 1995, makes it simpler to synthesize MA, uses less strictly controlled ingredients, produces less odor than chemical reactions involving P2P, and yields a more potent and psychoactive form of MA (with a higher percentage of the more active dextro steroisomer, rather than equal proportions of dextro and levo stereoisomers produced by the P2P method) (Burton, 1991; Cho, 1990; CSAT, 1997; Drug Enforcement Agency [DEA], 1996). In addition, dextro-MA is three to four times more potent to the central nervous system than levo-MA (Sowder and Beschner, 1993). Therefore, the MA currently being manufactured has especially potent effects.

Illicit MA is also likely to contain potentially toxic contaminants from unintended reaction byproducts and reagent residuals as well as processing errors. Many clandestine laboratories are operated by uneducated and unskilled chemists who get recipes from unpublished, handwritten sources or through the Internet. As with cocaine, most of the contaminants are intentional fillers used to dilute or cut the product and may include lactose, lidocaine, procaine, caffeine, quinine, or sodium bicarbonate.

Other impurities in illicit MA can cause dangerous toxic reactions. Some identified contaminants have been shown to have great potency for producing seizures in mice. Poisoning from other reagents and organic byproducts, including mercury, has also been suspected but not documented (Burton, 1991).

Patterns of Use

The effects of stimulant use also reflect the temporal pattern of drug administration and the user's experience history or chronicity. Users describe various motivations for initial experimentation with cocaine or MA, including a desire for heightening a sense of well-being and euphoria, increasing alertness and energy, boosting self-esteem, enhancing sexual desire and responsiveness, dispelling fatigue, improving performance, losing weight, or consuming more alcohol without feeling intoxicated (Hando and Hall, 1997; Sowder and Beschner, 1993). Some users only administer stimulants periodically, although most discover that tolerance builds rapidly to many of the desired effects, particularly euphoria, so that increasing doses are needed to achieve similar effects.

Although serious medical, psychological, and social consequences have followed experimental low-dose use of stimulants, two other patterns of self-administration are of greater concern. The fourth edition of *The Diagnostic and Statistical Manual of Mental Disorders* (DSM-IV) (American Psychiatric Press, 1994) characterizes these as (1) episodic use, separated by at least 2 days of nonuse, with gradually escalating doses and more potent routes of administration that often culminate in binge use; and (2) daily, or almost daily, use with no wide fluctuations in dose, but a gradual escalation. Actually, compulsive users probably represent only 5 to 10 percent of the total number of MA users and an even smaller percentage of all users of amphetamines (Cho, 1990).

Because of their different pharmacological properties, MA users typically administer the drug on a daily basis, whereas crack cocaine users binge on large amounts for a shorter period, interspersed by periods of nonuse (CSAT, 1997; King and Ellinwood, 1997). Figure 5-4 illustrates rapidly escalating and sustained plasma levels attained by different time patterns of drug administration during cocaine and MA binges. An extremely compulsive pattern of rapidly repeated injections or inhalations is required to sustain high plasma levels of cocaine, in contrast to amphetamine/MA, which has a longer half-life (Ellinwood and Lee, 1989).

The greatest behavioral pathology and most serious medical consequences usually follow compulsive bingeing with high doses of either smoked or injected MA or cocaine (Ellinwood and Lee, 1989). The following paragraphs describe the sequence of phases that typically occur in the establishment of this hazardous, "high-transition" pattern and some of their accompanying side effects (as depicted in Ellinwood and Lee, 1989; King and Ellinwood, 1997). Knowledge of these phases can be useful for the medical practitioner in taking a substance use history and understanding what effects are likely to accompany a particular stage of acute intoxication, withdrawal, or more chronic use patterns.

Intoxication

Stimulant use phases

- **Initiation, single-dose phase**. Early use of a single dose of stimulants results in euphoria and increased energy that correspond closely to stimulant plasma levels. Higher levels of euphoria are achieved by injection or inhalation routes of administration that evoke a rapid rise to peak drug concentrations. The rush experienced by persons who inhale (smoke) or inject stimulants is profoundly rewarding and reinforcing. Classical conditioning to the cues associated with drug use reputedly occurs during this initial phase.
- **Consolidation, dose-frequency escalation phase**. As tolerance develops to the

euphorigenic effects, users tend to increase doses and frequency of stimulant administration in an attempt to recapture the original and most intense rush sensations. They may also switch the route of administration to get a more rapid response. During this phase, intermittent consumption is prolonged with the discovery that higher doses produce greater effects.

- **Maintenance phase with bingeing**. High-dose and frequent-use patterns often lead to even more compulsive bingeing over a few hours to days that ceases only when the user is totally exhausted or the stimulant supply runs out. Binges typically last 12 to 18 hours (but may last 2 to 3 days or longer) for cocaine users and much longer—from 3 to 15 days—for MA users. The high and sustained plasma levels achieved during binges can have considerable pathological effects. The binge is characterized by frequent mood swings as plasma levels of the stimulant fluctuate. Stereotypic behaviors and thinking exclude other concerns so that the user focuses exclusively on internal sensations and withdraws from social activities in pursuit of direct pharmacological effects. Almost all activity is directed to acquiring the drug and consuming it. Also, the settings in which drugs are consumed are progressively restricted.

"Crash" and withdrawal syndrome phases

- **Early crash phase**. The binge terminates with a "crash" that has several successive phases that follow each other in rapid succession over a relatively brief interval after a cocaine run, but are more prolonged and pronounced in MA use. Dysphoria, anxiety, and agitation begin a short time after cessation of stimulant use, followed by an intense drug craving that often leads to recidivism. Users may exhibit a repetitive cycle of bingeing, with an intervening crash, over a period of several months. The more protracted withdrawal from ice produces a particularly irritable and jittery state that coincides with the initial "come-down" period after binge use and is a dangerous time because the user is short-tempered and unpredictable. This "tweaking" period is exacerbated by the user's prolonged lack of sleep. At this point, the tweaker is extremely frustrated because no dose will reestablish euphoria. Although not apparently intoxicated, the tweaker may have rapid eye movements, concise but quivery speech, and brisk, somewhat jerky movements The tweaker's thinking seems scattered and subject to paranoid delusions.
- **Middle crash phase**. Anxiety and agitation are followed by a period of fatigue, increasing depression, and anhedonia with decreased mental and physical energy. An intense desire for sleep, often accompanied by insomnia, usually replaces the drug craving. During this part of the crash, users may use alcohol, benzodiazepines, or opiates to induce and prolong sleep. The middle crash phase terminates with a period of protracted sleep, often for 24 to 36 hours during which time any attempt at therapy or other intervention is inappropriate.
- **Late crash phase**. The period of hypersomnolence in the late crash phase is often followed upon wakening by intense hunger.
- **Protracted withdrawal**. Following the crash phase (or early withdrawal), the user experiences symptoms that are opposite to those of stimulant intoxication: fatigue, loss of physical and mental energy, depression, anhedonia, and a limited interest in his surroundings. These symptoms may increase in intensity over the 12 to 96 hours immediately following the crash, or they may wax and wane over several weeks. A severe and persisting depression in this

phase can result in suicidal ideation or suicide attempts and is a major concern for the user. Anhedonia and dysphoria usually dissipate over a 6- to 18-week period for MA users. In the protracted withdrawal phase, periods of drug craving may reemerge. These cravings are often triggered by conditioned environmental cues and can only be extinguished by sustained abstinence.

Tolerance/Sensitization To Stimulant Effects

Chronic users of stimulants develop tolerance to many of the initial effects, often after only a few weeks of drug use. This means that a higher dose is required to achieve the same effects, or markedly diminished effects are attained if the same dose is continued (American Psychiatric Press, 1994). Most notably, tolerance develops rapidly to the euphorogenic effects of stimulants and is the ostensible cause for most dose escalation by stimulant users—although dose increases may also stem from a desire to experience more intense effects. Tolerance also develops to the anorectic effects of MA in humans because weight loss stops after several weeks. Tolerance also appears to develop to the cardiotoxic effects of large doses of MA that many users survive. In fact, many of the initial symptoms of stimulant intoxication disappear with chronic use: Blood pressure may be normal, and nausea and vomiting are seldom seen. This tolerance is not the result of increased MA metabolism because chronic users show metabolic patterns similar to naive users (Angrist, 1994).

Interestingly, chronic, high-dose stimulant users may also become sensitized to the drug, a unique phenomenon characteristic of psychomotor stimulants. Sensitization is essentially the reverse of tolerance and produces undesirable effects with lower doses of the drug than were required to yield these same reactions in an earlier phase of the addiction process. There appears to be some sensitization to the psychosis-inducing effects of stimulants in humans. After one psychotic episode is experienced following chronic, high-dose use, a lower minimal dose of cocaine or MA may induce another psychotic episode, with more rapid onset following drug intake and a longer duration than the initial psychosis. Many sensitized stimulant users experience an almost immediate return of paranoia, psychosis, and stereotyped thinking if drug use is resumed (Angrist, 1994; CSAT, 1997). The sensitization process in stimulant dependence is elaborated in the section on toxic psychosis in this chapter.

Clinical Manifestations And Medical Management

As already noted, the intensity and duration of acute manifestations of stimulant intoxication correlate generally with the rate of rise and the height of peak blood levels reflected in brain concentrations. Acute intoxication with stimulants resembles hypomania or a manic state. In low doses, the libido is stimulated and sexual performance is enhanced. In high doses, spontaneous ejaculation and orgasm can occur. With increasing doses, poor judgment, indiscretions, sexual acting-out, and other bizarre behaviors or mental alterations are more likely to be seen. Acute stimulant intoxication can result in seizures, confusion, dystonias, respiratory depression, chest pain, or cardiac arrhythmias (Gold and Miller, 1997) (see Figure 5-4).

Distinctive Characteristics Of MA Intoxication

- There may be a peculiar odor of ammonia or stale urine, especially among users who smoke MA that has been crudely synthesized

Figure 5-4
Common Signs and Symptoms of Acute Stimulant Intoxication

Physiological	Psychological/Behavioral
■ Dilated pupils ■ Diaphoresis (profuse sweating)—often with chills ■ Hypertension (elevated blood pressure) ■ Tachycardia (increased heartbeat), with or without arrhythmia and chest pain ■ Bradycardia (slowed heart action) ■ Hyperthermia (elevated temperature) ■ Suppressed appetite, weight loss ■ Bruxism (teeth grinding) ■ Insomnia or decreased need for sleep ■ Tremors ■ Seizures—mostly for cocaine users ■ Headache—occasionally	■ Euphoria, heightened sense of well being ■ Increased vigor, giddiness, and sense of enhanced mental acuity and performance ■ Agitation, restlessness, irritability ■ Garrulousness, with pressure of speech, flight of ideas, and rapid shifts in thinking ■ Poor concentration ■ Grandiosity, exaggerated self-esteem, egocentricity ■ Hypervigilance, with increased curiosity about the environment ■ Enhanced sensory awareness ■ Fearlessness, suspiciousness ■ Impaired judgment, poor impulse control ■ Clear sensorium, not usually disoriented ■ Aggression and emotional lability, with potential for violence

in illicit laboratories. Smoked ice is, however, essentially odorless.

- The MA user may present with tachycardia (rapid heart rate), although not usually accompanied by arrhythmia (irregular heartbeat). Compared with cocaine intoxication, MA intoxication causes fewer heart, pulmonary, and circulatory problems, especially for users of newer forms of ephedrine-based dextro-MA that stimulate the heart, lungs, and blood vessels to a lesser degree than older forms containing equal parts of levo- and dextro-MA (Inaba et al., 1993).
- MA users are more likely to appear in the ER as a result of trauma from fighting or motor vehicle accidents than for physical complaints.
- Because of its longer lasting effects, MA abuse may lead to more frequent psychiatric impairment, more potent central nervous system (CNS) effects, and more overdoses. Chronic abuse of MA (beyond 2 weeks) is more hazardous than chronic cocaine abuse because of MA's sustained effects. Moreover, drug-induced psychoses in MA users are likely to last longer than those of cocaine users and, in addition, may not respond as readily to available treatments.
- MA users are more likely than cocaine users to be single substance rather than polysubstance users (although many also use marijuana).
- Stereotyped activity—persistent, repetitive, and compulsive activity such as vacuuming the same part of the floor over and over again, popping knuckles repeatedly, picking at scabs, or taking apart and reassembling mechanical devices—may appear in MA users.

Distinctive Characteristics Of Cocaine Intoxication

- Cocaine users are more likely than MA users to present with serious and potentially lethal physical complications (e.g., cardiac arrhythmia, chest pains, cerebrovascular accident [e.g., strokes], toxic seizures, hypertension crises, hyperthermia).
- Cocaine users are also more likely than MA users to use multiple substances, especially alcohol, benzodiazepines, or opiates.

Management of Stimulant Intoxication

Acute MA intoxication, unless delirium or psychosis is present, seldom comes to medical attention. Most cocaine users who come to an ER with drug-related complaints have not used the drug for several hours, and peak plasma levels have already subsided, especially if the cocaine was injected or smoked (Rowbotham, 1993).

Uncomplicated intoxication requires only observation and monitoring in a subdued environment until symptoms subside over several hours. General measures include monitoring of vital signs for rising pulse rate, temperature, or blood pressure; providing a quiet and cool environment that helps to diminish agitation and overreaction to external stimuli; and close observation. Physical exertion and an overheated room can potentiate adverse effects because stimulants affect the body's heat-regulating mechanism at the same time that blood vessel constriction conserves heat. Although verbal reassurance is usually sufficient for quieting the patient, indications that agitation is escalating and moving toward paranoia and potential psychosis, with increasing risk for violence, may warrant pharmacological intervention. Fast-acting benzodiazepines such as lorazepam (Ativan) or diazepam (Valium) are useful for calming an anxious, agitated patient (Ellinwood, 1975; Weis, 1997).

Stimulant Overdose

Toxic, fatal, or subfatal syndromes are seldom seen in chronic, high-dose, intravenous stimulant users, probably because tolerance develops rapidly. Most stimulant overdose fatalities occur in neophytes or persons who accidentally ingest large amounts, such as "bodypackers" or children (Ellinwood, 1975). (Bodypackers are individuals who have swallowed waterproof packets filled with cocaine, usually in South America, in an attempt to clear U.S. customs undetected and then pass the packets through the gastrointestinal tract.) It should be noted, however, that the toxic dose for stimulants has enormous variability and appears to be idiosyncratic and unpredictable, without a known relationship to body weight. Hence, the amount of cocaine or MA used is not a reliable predictor of the reaction (Weis, 1997).

The symptoms of a sublethal stimulant overdose may include dizziness, tremor, irritability, confusion, hostility, hallucinations, panic, headache, skin flushing, chest pain, palpitations, cardiac arrhythmias, hypertension, vomiting, cramps, and excessive sweating. High doses of stimulants may cause high fever, cardiac arrhythmias and arrest, irregular breathing, seizures, and stroke. Agitated states characterized by increased aggressiveness or psychotic features may also occur with intoxication, particularly for MA (Weis, 1997). The development of hyperpyrexia (excessively high fever), severe hypertension, convulsions, and cardiovascular collapse signal a life-threatening situation (Ellinwood, 1975).

Lethal doses of stimulants—administered to laboratory dogs by injection or observed in bodypackers when ingested packets leak or break—produce a predictable sequence of events culminating in generalized convulsions and death. Heart rate, blood pressure, cardiac output, and body temperature rise rapidly, and a toxic delirium is observed before generalized and terminal seizures begin (Ellinwood, 1975; Rowbotham, 1993; Wetli, 1993).

Management of a Potentially Lethal Overdose

Stimulant users who present with life-threatening medical conditions and toxic drug levels should be treated with standard life-saving techniques that respond to the presenting symptoms (Weis, 1997). Acute neurological symptoms such as seizures or rapidly elevating vital signs require immediate intervention. Nondrug causes of any symptoms should be carefully ruled out, and the patient should also be evaluated for multiple substance use. Stimulant overdose patients should be hospitalized, especially if treatment is threatened by polysubstance use (Gold, 1997).

No specific antidotes or antagonists to stimulant overdose are available—unlike naloxone (Narcan) for opiates and the benzodiazepine antagonist flumazenil (Romazicon). However, the following procedures are suggested:

- Request specialist consultations as needed.
- Manage hyperthermia by sedating to slow down and stop agitated movements and rapidly cooling the patient with body ice packs, mist and fan techniques, or cooling blankets (Ellinwood, 1975; Gold, 1997). Simple measures such as preventing or correcting elevated body temperature can be remarkably effective in preventing death from cocaine toxicity (Rowbotham, 1993). Although dantrolene (Dantrium) can be administered for very serious and escalating, uncontrolled hyperthermia (Weis, 1997), such drugs do not always enhance the cooling process for patients with life-threatening hyperthermia (Goldfrank and Hoffman, 1993).
- If restraints are required to start an intravenous administration, use mesh-type blankets only transiently to avoid interfering further with heat loss.
- Provide adequate ventilation and oxygenation.
- Uncontrolled hypertension can be managed by intravenous administration of phentolamine (Regitine) or dopamine (Intropin). Although clinical experience supports the use of labetalol (Normodyne) for treating hypertension, no controlled experimental studies support the efficacy of this drug as an alpha-adrenergic blocker (its beta-adrenergic effects are more potent). Rapidly acting and easily controlled antihypertensive agents such as the vasodilator nitroprusside or the alpha-adrenergic blocker phentolamine are usually preferable (Goldfrank and Hoffman, 1993).
- Treat seizures like status epilepticus with intravenous diazepam or other benzodiazepine. Diazepam is most effective if administered before or shortly after cocaine ingestion but is less effective after seizures begin (Rowbotham, 1993). Phenobarbitol or phenytoin (Dilantin) may be used if diazepam is ineffective (Schrank, 1993). Alternatively, 25 to 50 mg of intravenous pentobarbitol can be administered to control cocaine-induced seizures (Gold, 1997).
- Complaints of chest pain warrant evaluation for possible myocardial ischemia and infarction. Nitrates are indicated for cocaine-induced myocardial ischemia to alleviate coronary vasoconstriction. Beta-adrenergic blockers such as propranolol (Inderal) should not be used because they may enhance vasospasm. Aspirin should be administered, unless contraindicated, to reduce cocaine-mediated platelet aggregation (Goldfrank and Hoffman, 1993).

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Use standard treatments for arrhythmias, including phenytoin. Atrial arrhythmias that do not respond to cooling and sedation may require cautious use of calcium channel blockers or mixed alpha-/beta-adrenergic blockers such as verapamil (Calan), esmolol (Brevibloc), and labetalol (Goldfrank and Hoffman, 1993). Lidocaine may be contraindicated for ventricular arrhythmias that begin immediately after cocaine use as a response to catecholamine excess but is appropriate for ventricular arrhythmias that indicate an ischemic myocardium. Sodium bicarbonate has proven useful for cocaine-induced wide-complex arrhythmias (Goldfrank and Hoffman, 1993). Also note that management of acute psychiatric manifestations of cocaine intoxication by sedation appears to have a salutary effect on emerging cardiovascular complications.

In general, phenothiazines, especially chlorpromazine (Thorazine, Mellerial), are contraindicated because these drugs lower the seizure threshold (Gold, 1997). Haloperidol (Haldol) has not proven efficacious in preclinical studies in protecting against cocaine-induced seizures or fatalities, but it may have utility for MA-induced psychoses. The serious difficulties encountered in using haloperidol for sedative–hypnotic withdrawal in humans when agitation and hyperthermia are present may also apply to its use for acutely agitated or psychotic stimulant users who already have deficits in thermoregulatory control. Haloperidol may precipitate or exacerbate acute dystonic reactions associated with cocaine use (Goldfrank and Hoffman, 1993).

Manifestations of Stimulant Withdrawal/Abstinence

A characteristic withdrawal-type syndrome usually develops within hours to days after cessation of prolonged and heavy stimulant use. The symptoms can follow long-term use or much shorter binges. Although "cocaine blues" were described as early as the turn of the century (Gawin and Kleber, 1986), more recent investigators note that stimulant withdrawal is much less definitive than withdrawal from opiates or alcohol and has not been so well studied (Lago and Kosten, 1994; West and Gossop, 1994). (See Figure 5-5 for some common signs of stimulant withdrawal.)

Some clinicians distinguish between stimulant withdrawal symptoms following a several-day binge and complaints that characterize withdrawal after more chronic high-dose use. Stimulant users who have binged for 2 to 3 days are dysphoric, exhausted, and sleep for 24 to 48 hours. Cocaine users in this category commonly use alcohol, marijuana, benzodiazepines, or heroin with cocaine to reduce irritability. Following more chronic and regular stimulant use, withdrawal symptoms that subside over 2 to 4 days include dysphoria, irritability, difficulty sleeping, and intense dreaming (CSAT, 1995d).

Other clinicians emphasize differences in severity between withdrawal from cocaine and withdrawal from MA. A substantial number of persons with cocaine dependence have no clinically evident withdrawal symptoms. For the minority of cocaine users who do complain, symptoms begin within hours to days of the last dose, the crash lasts for 3 to 4 days, withdrawal persists from 1 to 10 weeks, with waxing and waning of the drug craving. The mood state of the cocaine user may return to normal after several days to a month.

Withdrawal symptoms seem to be most severe in the initial days following cessation of use (Cornish and O'Brien, 1996; Gold and Miller, 1997). Although there are no physical manifestations of a withdrawal syndrome when MA use is stopped, there are several symptoms that occur when a chronic user stops taking the drug (National Institute on Drug Abuse [NIDA], 1998a). Symptoms begin 12 to 24 hours after binge use is terminated and may persist for 1 to

Figure 5-5

Common Signs and Symptoms of Stimulant Withdrawal/Abstinence Syndrome

Physiological	**Psychological/Behavioral**
■ Thin, gaunt appearance with reported weight loss or anorexia ■ Dehydration ■ Fatigue and lassitude, with lack of mental or physical energy ■ Dulled sensorium ■ Psychomotor lethargy and retardation—may be preceded by agitation ■ Hunger ■ Chills ■ Insomnia followed by hypersomnia	■ Dysphoric mood—that may deepen into clinical depression and suicidal ideation ■ Persistent and intense drug craving ■ Anxiety and irritability ■ Impaired memory ■ Anhedonia—loss of interest in pleasurable activities ■ Interpersonal withdrawal ■ Intense and vivid drug-related dreams

2 weeks. The client initially feels depressed and anxious, with an intense craving for MA.

This phase is followed by fatigue and sleepiness, although this may be mixed with insomnia. Upon awakening after prolonged sleep, the client may be very hungry, and there may be persisting anhedonia and dysphoria. Other symptoms include paranoia and aggression. Depression appears to be more severe and prolonged following MA use and is correlated with duration of use and size of the doses (Gold and Miller, 1997; Gawin and Ellinwood, 1988).

Management of Stimulant Withdrawal

Stimulant withdrawal is not medically life threatening and, unlike alcohol or barbiturate withdrawal, does not require pharmaceutical intervention. Although no consistent physiological disruptions requiring gradual withdrawal have been observed, some medications may be used to attenuate symptoms and provide support.

The greatest risk from the distinctive stimulant abstinence syndrome is of doing harm to self or others. Because withdrawal-related dysphoria and depression can be particularly severe in stimulant users, risk of suicide is intensified, and sensitive management is essential. Cocaine-induced depression usually dissipates fairly rapidly—in a matter of hours. The depression is agitated and often related to actual situations resulting from drug use (e.g., the client is disturbed that all of his money has been "blown" on the cocaine binge or that interpersonal relationships are jeopardized by his continuing drug dependence).

However, withdrawal-associated depression following high-dose MA use is more prolonged. During the tweaking phase of withdrawal, the high-dose MA user begins a rocky, jittery reaction characterized by agitated paranoia, extreme frustration, and the return of intense drug cravings. Suicidal ideation may be high, and violence is easily provoked.

Tweaking effects after persistent bingeing on ice are particularly dangerous. Clients may misinterpret caretakers' gestures and turn against them. Restraints and sedation in a secure facility may be necessary. Although stress reduction techniques and other approaches to preventing harm should be used standardly, medical personnel can also use benzodiazepines (e.g., diazepam) to control agitation and tachycardia (see further discussion of violence as a special issue).

For clients with preexisting diagnosed or unrecognized clinical depression, cocaine worsens symptomatology. These individuals are most likely to experience deepening dysphoria and/or paranoia after cocaine use. Treatment with selective serotonin reuptake inhibitors (SSRIs) may be of use (Gold, 1997).

Continuing agitation and persistent inability to fall asleep during the tweaking stage may also be treated symptomatically by using the antidepressant trazodone (Desyrel), whose dopaminergic properties help to sedate the client. Benadryl is also used for its sedating properties and for its effects on the dermatologic problems that often accompany MA use (e.g., itching and hypersensitivity of the skin). However, caution should be exercised in using any medications with high abuse/dependence potential. In general, prescriptions should not be written for use outside the treatment facility because use or resale of these drugs is very tempting to this population.

After the tweaking stage, MA abstainers usually "crash" and sleep several days at a time, depending on the dose and duration of the binge. This hypersomnolence may interfere with assessment of mental status and potential for dangerous behavior. Hence, clients should be evaluated immediately after wakening from this prolonged sleep for persisting dysphoria and other psychiatric symptoms of anxiety and depression (Weis, 1997). During this hypersomnolent state, and until sleep deprivation is overcome, active participation in therapy or followup of a referral to a treatment program by stimulant users is not a realistic expectation.

Drug craving during stimulant withdrawal has been treated with a variety of medications (e.g., bromocriptine, amantadine) without demonstrated efficacy in alleviating symptoms, getting clients "clean," or preventing relapse.

"Cocaine dreams" may occur during this period or as late as 8 or 9 months after termination of stimulant use during a protracted abstinence phase. They usually entail vivid recall of actually using and experiencing the high. The client may actually sweat and experience other symptoms of intoxication while dreaming. These intense dreams, which may sometimes contain vignettes in which the drug user loses or drops a supply or refuses to smoke crack/ice, can be used therapeutically to convince clients that they are making progress in treatment by making a subconscious choice not to use. Otherwise, the dreams may enhance drug cravings and intensify a vulnerability for relapse. These dreams are primarily experienced by users of injected cocaine/MA and smoked crack or ice.

Because stimulant users frequently self-medicate withdrawal symptoms with alcohol, benzodiazepines, or opiates, there may be symptoms of withdrawal from these drugs if they have been used continuously or at high doses. These require specific management and titration of substitute doses or other means of alleviating symptoms.

Manifestations of Chronic Stimulant Use Disorders

Although fatalities from stimulant overdose or acute myocardial infarction following administration of cocaine by inexperienced users have been documented, and other medical and psychiatric complications have been observed at all dose levels and routes of administration among naïve users, the majority of serious stimulant-induced medical and psychological complications follows chronic, high-dose use.

Because tolerance develops rapidly to the subjective and cardiovascular effects of stimulants, the specification of complications following chronic use is complex (Rowbotham, 1993). However, cocaine toxicity affects nearly every organ system, with the most dramatic changes found in the cardiovascular system, the brain, the liver, and the pulmonary system (Majewska, 1996). Although there are some minor differences between the sequelae of chronic MA and cocaine use, the incidence of such side effects as chest pain, seizures, paranoid reactions, and suicidal thoughts is about the same for both drugs. Chronic MA users appear to have more headaches, severe depression, and hallucinations than counterpart cocaine users, but the evidence from community samples is not definitive (CSAT, 1997).

Figure 5-6 summarizes some of the more common symptoms and potentially serious complaints presented by chronic stimulant users. The following section contains a detailed description of stimulant-induced medical and psychiatric complications with limited comments about management of these conditions. Schrank specifies more detailed ER procedures for responding to some of the more frequently seen complications (Schrank, 1993). Figure 5-7 shows the distinctive indicators of chronic MA use and chronic cocaine use.

Identification and Management of Medical Complications

Cardiovascular System Effects

Cardiotoxicity stemming from catecholamine excess is observed in both cocaine and MA users. Although the cardiac effects are more profound for MA users because this drug results in even greater elevation of catecholamines than cocaine, the incidence of fatalities following myocardial infarction has been less frequent until the recent increase in inhaling (smoking) or injecting MA (Cho, 1990; Cook et al., 1993; CSAT, 1997).

Stimulants, especially cocaine, have been linked to virtually every form of heart disease, including different forms of arrhythmias, coronary vasospasm, myocardial ischemia, myocardial infarction, and cardiomyopathy (Cornish and O'Brien, 1996; Gold, 1997). Case reports of fatalities from myocardial infarction and tachyarrhythmias document their occurrence at all dose levels and routes of administration in otherwise healthy young adults without the usual coronary risk factors, but preexisting coronary artery disease can exacerbate the response and increase the likelihood of sudden death, as can hyperthermia and agitation (Ellinwood and Lee, 1989; Gold, 1997; Schrank, 1993). Tachycardia, hypertension, ruptured blood vessels, arrhythmias, and arteriosclerotic lesions typically precede myocardial ischemia and infarction (Majewska, 1996). With prompt medical intervention, patients generally survive stimulant-induced cardiomyopathy with heart failure (CSAT, 1997). There is some controversy about optimal pharmacologic approaches to treating arrhythmias and other cardiac effects. Lidocaine was previously used but may be contraindicated for ventricular arrhythmias because it lowers the seizure threshold (Goldfrank and Hoffman, 1993). However, certain calcium channel blockers (e.g., Nifedipine, diltiazem, verapamil) seem promising (Gold, 1997; Schrank, 1993).

Respiratory/Pulmonary Effects

Cocaine crack smokers frequently seek medical attention for difficulties in breathing (dyspnea) or severe chest pain. This may result from cocaine-induced pulmonary hemorrhage, lung damage, pneumonia, pulmonary edema, asthma, pneumothorax, pneumomediastinum, or pneumopericardium (Cornish and O'Brien, 1996; Gold, 1997). Pulmonary barotrauma may

Figure 5-6

Common Symptoms of Chronic Stimulant Abuse/Dependence

Physiological	**Psychological/Behavioral**
■ Extreme fatigue—with physical and mental exhaustion and disrupted sleep patterns ■ Nutritional disorders—extreme weight loss, anemia, anorexia, cachexia (body wasting) ■ Poor hygiene and self-care ■ Skin disorders and secondary skin infections—itching, lesions, hives, urticaria ■ Hair loss ■ Muscle pain/tenderness—may indicate rhabdomyolysis ■ Cardiovascular damage—from toxicity and contaminants in MA production, with concomitant renal and hepatic problems ■ Hypertensive crises with renal damage from sustained hypertension ■ Difficulty breathing—may reflect pulmonary edema, pneumonitis, obstructive airway disease, barotrauma, and other complications ■ Myocarditis, infarcts ■ Headaches, strokes, seizures, vision loss ■ Choreoathetoid (involuntary movement) disorders ■ Impaired sexual performance and reproductive functioning ■ Cerebrovascular changes, including evidence of cerebral hemorrhages and atrophy with associated cognitive deficits ■ Ischemic bowel, gastrointestinal complaints	■ Paranoia with misinterpretation of environmental cues; psychosis with delusions, and hallucinations ■ Apprehension—with hopelessness and a fear of impending doom that resembles panic disorder ■ Depression—with suicidal thinking and behavior ■ Acute anxiety ■ Eating disorders

result from spasmic coughing following smoke inhalation or odd mechanisms of drug delivery (mouth-to-mouth inhalation), with sudden increases in airway pressure that result in alveolar rupture and the induction of free air into the pleural cavity, mediastinum, or subcutaneous tissues. The amount of free air is usually small and resolves spontaneously under observation. The possibility of esophageal rupture should be considered in pneumomediastinum if vomiting is present (Schrank, 1993). Cocaine can also cause sudden death by respiratory failure from drug-induced inhibition of the medullary centers in the brain (Gold, 1997).

Tracheobronchitis with cough is a frequent accompaniment of crack smoking, as are lobar and nonlobar pneumonias. Bronchospasm is another complaint of these smokers, usually in clients with a history of asthma (Schrank, 1993). Crack lung is a new syndrome that manifests with symptoms of pneumonia—severe chest pains and breathing problems with high fever—but no substantiating lung X-ray evidence. The condition does not respond to standard treatment, although anti-inflammatory drugs may relieve symptoms. Clients with crack lung may suffer from oxygen starvation or loss of blood with potentially fatal results (Gold, 1997).

Pulmonary edema has been observed in both cocaine and MA fatalities and attributed variously to deep inhalation aggravation of preexisting conditions (Nestor et al., 1989) and granulomas formed in response to adulterants added to the drugs (CSAT, 1997). Chronic obstructive lung disease in MA users is thought to result from thrombosis of small pulmonary vessels with gradual reduction of pulmonary vascular bed, pulmonary fibrosis, and granuloma formation (CSAT, 1997).

Cerebrovascular Complications

An increasing amount of research has recently focused on neurological impairments apparently resulting from high-dose and chronic use of stimulants, particularly by more rapid routes of administration. Some of the more devastating cerebrovascular consequences of cocaine and MA use have been known for years—seizures, ischemic strokes, and subarachnoid and intracerebral hemorrhages. Other neurological complications include optic neuropathy, global brain ischemia, and edema following myocardial infarction. Newer brain-imaging techniques now demonstrate various degrees of previously undetected and unsuspected cerebral atrophy and brain lesions in many chronic cocaine users (Cornish and O'Brien, 1996; Schrank, 1993; Majewska, 1996).

Seizures are a well-known complication of cocaine use, occurring almost immediately after any of the more rapid delivery routes, but not always dose-related. Chronic use may sensitize (kindle) an individual's response, but this is not definitively proven (Daras, 1996; Gold, 1997). Most cocaine-induced seizures are of short duration and leave no residual effects, although prolonged seizures can be catastrophic (see earlier references under overdose management) (Schrank, 1993; Cornish and O'Brien, 1996).

Cerebral hemorrhage and ischemic strokes are relatively rare events for stimulant users but

Figure 5-7

Distinctive Indicators of Chronic Abuse of Cocaine Versus MA

Cocaine	MA
▪ Nasal perforations and nose bleeds among snorters ▪ Serious constipation due to dehydration and insufficient dietary fiber	▪ Dental problems, including missing teeth, bleeding and infected gums, dental caries ▪ Muscle cramping related to dehydration, with low magnesium and potassium levels ▪ Dermatitis around the mouth from smoking hydrochloride salt ▪ Stale urine smell due to ammonia constituents used in manufacturing MA ▪ Various dermatologic conditions, including excoriated skin lesions

occur more frequently with users of crack and ice. At least half of those who suffer brain hemorrhages have such underlying abnormalities as arteriovenous malformations and cerebral aneurysms. Stimulant-induced hypertension probably leads to rupture of these abnormalities that will also require surgical intervention to correct. Cocaine-induced hypertension and vasospasm seem to be associated with other cases.

The toxic role of simultaneous alcohol and cocaine use that produces cocaethylene (see later discussion) is also under investigation (Schrank, 1993; Daras, 1996). Another unresolved issue is whether CNS vasculitis is a causal factor; MA-induced necrotizing vasculitis has been documented since 1970 (Miller et al., 1993). Cocaine-using clients who complain of headache while intoxicated should be evaluated for possible intracranial hemorrhage (Ellinwood and Lee, 1989; Daras, 1996).

The recently documented neurological deficiencies found in chronic cocaine users, particularly in the basal ganglia and frontal cortex, are similar to those found in a variety of neurological/psychiatric disorders, including bipolar disorder, schizophrenia, and frontal lobe degeneration from seizures, stroke, or injury that is accompanied by dementia, apathy, depression, and social disinhibition (Majewska, 1996). Animal studies have confirmed similar enduring, possibly permanent, CNS changes, associated with repeated high doses of MA. Neurotoxicity at an early age may predispose stimulant users to premature onset of movement disorders such as Parkinson's disease and other dystonic or choreoathetoid disorders involving undulating, involuntary, whole body movements that may appear at the end of a prolonged binge in chronic users and are not related to use of neuroleptic medications (CSAT, 1997; Gold and Miller, 1997).

An array of cognitive deficits is also observed in cocaine and other stimulant users that also characterizes brain aging and dementia and may indicate premature brain aging or possible cerebral atrophy in these drug-dependent individuals. These include problems in attention, concentration, problem-solving, abstraction, arithmetic performance, new learning, and short-term memory (Majewska, 1996; Cornish and O'Brien, 1996; Gold, 1997). Unfortunately, many of the studies documenting these deficits lack adequate data on respondents' premorbid performance (Daras, 1996).

Muscular and Renal Toxicity

Cocaine and MA may be direct muscle toxins because acute rhabdomyolysis—a condition that destroys skeletal muscle—has been diagnosed in users who did not have any of the previously associated risk factors (i.e., hyperthermia, agitation, seizures, hypotension, toxic delirium or coma, or acute renal failure). Muscle necrosis may occur after any route of drug administration, and the presence of rhabdomyolysis should be considered in stimulant-intoxicated clients, particularly those complaining of myalgia or muscle tenderness. One study found that one-fourth of clients presenting with cocaine-related complaints had evidence of mild, usually asymptomatic, rhabdomyolysis—defined as elevated creatine kinase levels that were five times higher than normal (Goldfrank and Hoffman, 1993; Schrank, 1993).

Although mild cases of rhabdomyolysis may not lead to renal complications, renal insult and failure is a distinct possibility for patients with concomitant hyperthermia, seizures, delirium, or coma (Schrank, 1993). Renal failure from rhabdomyolysis has been reported as an outcome of cocaine and MA use (Scandling and Spital, 1982). Accompanying hepatic damage is rare and probably an idiosyncratic response (CSAT, 1997).

Gastrointestinal Complaints

Abdominal pain, nausea, and vomiting are experienced by some stimulant users, probably indicating mild intestinal ischemia. Severe bowel infarction with elevated white blood cell counts, metabolic acidosis, and shock have also been observed. Occasionally, severe abdominal pain, bowel obstruction, or sudden onset of seizures are indicative of leakage or rupture of packets of cocaine ingested by "bodypackers" (Schrank, 1993). Another observed syndrome, "cocaine colitis," manifests as abdominal pain along with diarrhea and bloody stools, probably indicating diffuse gastrointestinal hemorrhage and tissue necrosis (Goldfrank and Hoffman, 1993).

Infections

As already noted, intravenous injection of cocaine or MA is associated with a variety of infectious diseases. Unsterile paraphernalia are particularly likely to result in blood-borne transmission of HIV/AIDS and hepatitis B, C, and D. Associated malnutrition in chronic users further lowers resistance to infection. Injection cocaine users are at greater risk of infectious endocarditis than other parenteral drug users (Daras, 1996).

Disinhibition and the initial aphrodisiac effects of stimulants are associated with participation in high-risk and unprotected sexual activity. Vigorous and prolonged sexual activity or anal intercourse is likely to damage tissues or protective condoms and thereby increase the likelihood of transmitting sexually contracted diseases (Cornish and O'Brien, 1996).

Effects on Reproductive Function And Fetus/Newborn

Use of stimulants by pregnant women has been related to poor obstetrical outcomes and adverse effects for the developing fetus, the newborn and the older child. An increased incidence of preecalmpsia, spontaneous abortions, and *abruptio placentae* has been observed among cocaine-using pregnant women. Toxic effects also result in fetal cerebral infarctions as well as low birth weight for gestational age and small head circumference. Recent studies in several separate locations have found similar rates of these complications in MA- and cocaine-using women and their prenatally exposed offspring (CSAT, 1997; Oro and Dixon, 1987).

Newborns exposed to stimulants in the womb may have poor feeding and sleep patterns, tremor, and hypertonia. Difficulties in consoling these "jittery" babies may inhibit close bonding with their mothers and contribute to developmental problems (Gold, 1997). Despite a spate of articles outlining probable behavioral and cognitive deficiencies in prenatally stimulant-exposed children, more recent meta-analyses have only confirmed lower birth weight compared with controls, but no clearly attributable effects on the fetus, infant, or child (Rabin and Little, 1994; Cornish and O'Brien, 1996).

A major problem with most of the earlier studies is that polysubstance use among pregnant women is ubiquitous, so that attribution of prenatal-exposure effects to any single drug is very difficult. Additional methodological confounds were introduced by differences in mothers' nutritional status, prenatal care, socioeconomic level, and trimester of maternal drug use, as well as a failure to account for the impact of the home environment in predicting IQ (CSAT, 1997; Cornish and O'Brien, 1996). Essentially, the long-term effects of maternal stimulant use on offspring are not known.

Chronic, high-dose stimulant use also affects reproductive and sexual functioning in both males and females. Men report gynecomastia (development of breasts), loss of sexual interest, impotence, and difficulty in maintaining an erection or ejaculating. Women have derangements of the menstrual cycle, including amenorrhea and infertility, as well as greater difficulty in achieving orgasm (Gold, 1997). Female stimulant users who have irregular or no menstrual periods often think they cannot get pregnant, although they are not always infertile and can have unwanted pregnancies. Testing for pregnancy and regular use of birth control should be encouraged.

HIV/AIDS and Hepatitis

Increased HIV and hepatitis B and C transmission are likely consequences of increased stimulant use, particularly in individuals who inject intravenously and share equipment. Infection with HIV and other infectious diseases is spread among injection drug users primarily through the reuse of contaminated syringes, needles, or other paraphernalia by more than one person. In nearly one-third of Americans infected with HIV, injection drug use is a risk factor, making substance abuse the fastest growing vector for the spread of HIV in the nation (NIDA, 1998a).

Research also indicates that MA and related psychomotor stimulants can increase libido in users, in contrast to opiates, which actually decrease libido. However, long-term cocaine use may be associated with decreased sexual functioning, at least in men (Rawson et al., 1998b). In addition, the use of MA seems to be associated with rougher sex, which may lead to bleeding and abrasions. The combination of injection and sexual risks may result in HIV becoming a greater problem among MA users than among opiate and other substance users, something that already seems to be happening in California (NIDA, 1998a).

Identification and Management of Psychological Complications

Toxic Psychosis

Initially described by Young and Scoville in 1938, amphetamine psychosis is a usually brief and spontaneously remitting paranoid state that is frequently accompanied by intense, fear-evoking delusions and hallucinations, but with clear consciousness and a relatively intact formal thought process (Angrist, 1994). Stimulant-induced psychosis occurs while the user is intoxicated, not in withdrawal after drug cessation (Tinklenberg, 1975). The condition is not rare or idiosyncratic, but typically follows chronic, high-dose administration of amphetamines, MA, or cocaine. However, this drug-induced psychosis is more prevalent among amphetamine and MA users than those who use cocaine, probably because the short half-life of cocaine makes it difficult to accumulate and sustain high plasma levels of that drug (Angrist, 1994; King and Ellinwood, 1997). Nonetheless, the condition has been reported after acute intoxication in relatively naïve users and occasionally after low doses.

Original reports of the condition described a threshold dose for eliciting a psychotic response as chronic administration of 50 mg amphetamine daily, but at least 10 cases have been documented at lower doses, and there are also case studies of psychotic reactions after a single dose (usually high) or only brief exposure to the drug (Angrist, 1994). Because studies of stimulant users have found a surprising prevalence of coexisting, often premorbid, psychiatric disorders (one-fourth of participants in one study had preexisting schizophrenia), low doses of stimulants may actually precipitate latent schizophrenia in some users whose psychosis is then mistakenly diagnosed as stimulant-induced (Angrist, 1994).

Amphetamine-induced psychosis

Several investigators claim that a toxic paranoid reaction or psychosis, usually accompanied by delusions and/or hallucinations, is a probable complication of high-dose MA use. Amphetamine-induced psychosis has been investigated prospectively under experimental conditions in at least two studies involving fewer than 50 clients. Griffith and colleagues successfully elicited psychotic symptoms in 25 of 31 experienced users after high-dose administration of MA and observed that 22 of the 25 were frankly psychotic (Griffith et al., 1972). Bell evoked similar amphetamine-induced psychosis in 11 of 13 subjects (Bell, 1973)—the remaining 2 were found to have preexisting schizophrenia (Angrist, 1994). Surveys of chronic cocaine users in treatment have also found that one-half to two-thirds had paranoid experiences that were not trivial (Angrist, 1994).

However, there are methodological problems with each of the investigative approaches to studying stimulant-induced psychosis that make the findings less than compelling. When this condition is studied prospectively, the drug-experienced volunteers who must be used for ethical reasons have unknown (unobserved) sensitivity and tolerance to stimulants; the numbers are, of necessity, quite small for drawing definitive conclusions, and at least some of the participants have managed to continue drug use, even under rigorous laboratory conditions. Data from case reports have other drawbacks: The premorbid history of drug use and psychiatric status is unknown and may not be accurately reported by respondents (Angrist, 1994; CSAT, 1997).

Development of toxic psychosis

Some researchers and clinicians describe the development of stimulant-induced psychosis as an evolving process. Panel members depicted MA users as having brief and transient psychotic episodes before a full-blown psychosis emerges after more extensive chronic use. MA users often recognize these early psychotic effects and try to stave them off by self-medicating with alcohol or decreasing drug use. In several articles, Ellinwood and colleagues describe the evolution of MA-induced psychosis as progressively abnormal behaviors—beginning at moderately high doses—with intense feelings of curiosity about the environment and patterns of exploration that result, for example, in examining the punctuation periods in a magazine text for evidence of a secret code (Ellinwood et al., 1973).

This first enthusiasm about "discoveries" moves over time and increasing doses from "watching the world" to feelings of being watched. Behaviors become more fixed and stereotyped, culminating with intense suspiciousness and, in psychotic reactions, paranoid delusions that misinterpret environmental cues. Visual hallucinations may be overreactions to barely glimpsed and recognizable objects in the client's peripheral vision; auditory hallucinations similarly begin with hearing simple noises. In later stages of the psychosis, the client loses all contact with reality and has delusions of persecution. If he is exhausted after a prolonged binge, the hyper-reactivity to stimuli and confusion can lead to panic, sudden violence, even homicide (King and Ellinwood, 1997).

Manifestations of toxic psychosis

The DSM-IV (American Psychiatric Press, 1994) distinguishes between cocaine intoxication with "perceptual disturbances" and cocaine-induced psychotic disorder with either delusions or hallucinations (depending on which is the prominent feature). In the former, the drug user has intact reality testing and is aware that auditory, visual, or tactile hallucinations are substance-induced, not actual representations of external reality. Another common manifestation, before paranoia is rampant and as deterioration develops, is stereotypy—persistent, repetitive acts such as disassembling and reassembling radios or other small gadgets that seems to offer some relief from agitation and anxiety. Even though the client seems to know that the behavior is meaningless, stopping it results in irritability and frustration (King and Ellinwood, 1997; Tinklenberg, 1975). As chronic, high-dose stimulant consumption continues, most users also withdraw from all social interactions and initiate other bizarre behaviors before the intensive drug use culminates in paranoid reactions or psychosis without any insight into activities.

Symptoms of stimulant-induced toxic psychosis usually abate spontaneously within a week (CSAT, 1997). Hallucinations stop within 24 to 48 hours of abstinence, and paranoia and delusions decrease over the next week to 15 days. The client may sleep after the first 24 hours for as long as 3 days, with extensive dreaming during this phase (Ellinwood, 1975). Clinicians also report that drug-induced psychosis dissipates more quickly for cocaine users—usually in 1 to 3 days—compared with up to 2 to 3 weeks for MA users. Users of ice are reputed to have the most intense and persistent psychoses (Sowder and Beschner, 1993).

Toxic stimulant psychosis can have typical and atypical presentations. Case reviews have established that approximately 80 percent of these psychotic clients experience paranoid delusions; 60 to 70 percent have hallucinations; 12 percent have tactile hallucinations (e.g., cocaine bugs crawling on the skin); olfactory hallucinations are present in fewer than 10 percent; and about 7 percent become disoriented. Hyperactivity and excitation are usually present (Tinklenberg, 1975; Ellinwood, 1975; Angrist, 1994). The client is generally oriented and has intact memory and an appropriate level of consciousness. Clients remember the psychotic episodes with remarkable clarity (Tinklenberg, 1975; Ellinwood, 1975). Thought disorder, if present, is usually mild and transient (CSAT, 1997).

A few clients are confused—usually delirious from high doses to which they have not developed tolerance; bizarre, usually autoerotic, sexual behavior is present in some, and others have destructive outbursts or make unmotivated assaults. One investigator found no differences in symptoms between relatively naive and chronic amphetamine users with psychotic reactions, but others claim that individuals who binge on high doses over a few days have more delusions and disorganized hallucinations and paranoid ideation than chronic users who have more systematic delusions. Intravenous drug administration caused no change in symptoms, but more rapid progression to psychosis (Angrist, 1994; CSAT, 1997).

The role of drug sensitization

Several issues pertaining to stimulant-induced psychosis remain unresolved. There is some disagreement about the role of drug sensitization (kindling) in precipitating more frequent toxic psychotic reactions at smaller-than-previously-required doses and sooner after drug use is reinitiated following a period of abstinence. There is also disagreement about the role of sensitization in deepening the depression experienced after withdrawal. The mechanisms for this "reverse tolerance" are not fully understood. Although animal experiments have shown that daily, intermittent dosing with stimulants results in sensitization, studies of amphetamine-induced psychosis in humans have yielded more ambiguous results (CSAT, 1997). However, a 1991 survey by Satel and colleagues of 50 cocaine-dependent clients consecutively admitted to a treatment program found that two-thirds (68 percent) had experienced paranoid psychosis while intoxicated and during the immediate postdrug crash (Satel et al., 1991).

The reported characteristics of this paranoia were consistent with a sensitization process. All of those with a paranoid reaction had, on average, years of binge use before paranoid symptoms gradually emerged. Anxiety gradually intensified during binges before frank paranoid delusions were experienced. Once paranoia emerged, every subsequent binge produced intensified reactions (despite use of anxiolytic street drugs by half of the group to ameliorate paranoid reactions), and the onset of these delusions after starting a run accelerated over time. Half of those who had experienced paranoid psychosis acknowledged engaging in bizarre behavior such as hiding or compulsively "checking up" on things; nearly two-fifths had secured weapons to protect themselves from imagined assailants. This paranoia persisted for an average of 12 hours, with near total resolution in 97 percent of cases before awakening after the postbinge crash (Gawin and Khalsa-Denison, 1996).

Similar results are reported by Brady and colleagues after a 1991 study of another 55 cocaine-dependent clients in treatment—53 percent had experienced cocaine psychosis, most with less drug and increasing frequency over time and with more rapid onset for the majority (Brady et al., 1991). It seems clear that sensitization to the psychosis-inducing effects of cocaine does occur, although the evidence for sensitization with amphetamines is somewhat less clear (Angrist, 1994).

Stress-induced psychosis

The role of stress or other "triggers" such as alcohol use or insomnia in precipitating the return of psychotic symptoms that were initially induced by stimulants is also controversial. Some investigators have reported that stress can evoke the return of psychotic symptoms (delusions, hallucinations, paranoia, suicidal thoughts) without further amphetamine or MA use and after long periods of abstinence (NIDA, 1998a; Sowder and Beschner, 1993; Spotts and Spotts, 1980).

However, Angrist questions the accuracy of such reports of spontaneous or stress-induced psychosis following amphetamine psychosis because the reported cases were not carefully monitored with urine toxicologies to rule out continuing substance use or examined for the possibility of simultaneous development of another psychiatric disorder (Angrist, 1994).

Duration of toxic psychosis

The duration of toxic stimulant psychosis is another issue in some dispute. Typically, uncomplicated psychosis induced by stimulants resolves rapidly unless more of the drug is taken. However, several Japanese investigators (i.e., Tatesu, 1964; Nakatani, 1990; Iwanami et al., 1994 [as cited in Angrist, 1994]) have reported persisting psychoses in chronic stimulant users for up to 1 year after abstinence when amphetamine metabolites were no longer present.

Angrist argues that Western investigators do not see prolonged psychoses very frequently, and the persisting psychoses observed in Japan may actually be cases where stimulants precipitated latent schizophrenia (or bipolar disorder), or the disorder was present but undiagnosed before amphetamine use began (Angrist, 1994). He concludes that the potential for amphetamine to cause long-standing psychosis may be a complication for some individuals. This conclusion is, however, unproven because the premorbid state of clients in reported studies has not been known and because continued substance use has not been ruled out by urine toxicology monitoring.

Treatment of toxic psychosis

Treatment of the client who presents with toxic stimulant psychosis entails rapid, systematic visual assessment, continued observation and monitoring, and symptom management. All unnecessary stimulation should be reduced, but complete sensory deprivation should be avoided by providing quiet rooms with moderate lighting and sufficient space and insisting on subdued talking without any rapid or unexpected movements. The clinician should reassure the client that the condition is drug-induced and will subside (Tinklenberg, 1975). Restraints may be required initially to gain control of the client, but should be checked frequently to ensure that risk to extremities is minimized, that respiration is not compromised, and that heat loss is not inhibited. Agitation should be controlled promptly by sedation with parenteral benzodiazepines—usually diazepam.

Differential diagnosis of acute confusional states must be instituted immediately. Consideration should be given to the possibility of head injury, intracranial hemorrhage, or thyrotoxicosis. Information from significant others is helpful, and toxicology testing is also useful to confirm a diagnosis (Schrank, 1993).

Acute stimulant-induced psychosis should generally be managed in a hospital psychiatric department or similar facility. Minor psychotic episodes with low-grade symptoms that respond readily to neuroleptic medications may, on some occasions, be managed in a well-staffed, free-standing chemical dependency unit if sufficient personnel with training and experience in treating dual diagnosis are readily available. Urine testing is recommended to confirm a diagnosis of drug-induced psychosis because the syndrome can closely mimic other psychotic disorders such as schizophrenia, hypomania, depression, obsessive–compulsive reactions, or catatonia. However, a negative urine report does not necessarily mean that stimulants were not present (Ellinwood, 1975; Tinklenberg, 1975). The criteria for placement should reflect the persistence of the condition, the competence and training of personnel, and the drug taken. MA users who have accumulated high plasma levels from longer binges and larger doses of stimulants with longer half-lives are particularly prone to violence during psychosis—their paranoia makes them suspicious of attempts to medicate them, they are likely to become aggressive, and they don't comply with medication instructions after release from the hospital.

The criteria for continued hospitalization or inpatient care during psychosis are perceived risk or threats to self and others, as well as elevated vital signs, severe suicidal ideation, persistence of psychological or cognitive impairments beyond the usual time for spontaneous resolution, and severity of any medical problems such as serious heart disease, a history of infarcts, concomitant alcohol, barbiturate or opiate dependence, or diabetes and similar conditions that require careful monitoring (Tinklenberg, 1975). Release should not be considered until the medical crisis is resolved or until the patient has been stabilized psychologically for 24 hours and is able to self-calm without continuing use of neuroleptics. Buspirone hydrochloride (Buspar) is used experimentally in Hawaii to treat low-grade residual psychosis and is anecdotally effective when given in larger than usual doses (over 60 mg). It is relatively safe, although not advisable if the client has a history of benzodiazepine abuse.

Aggression and Violence

A major perceived problem associated with amphetamine/MA abuse is the potential for sudden and intense violence (Miczek and Tidey, 1989). MA has been associated with crime and combative behavior (Sowder and Beschner, 1993). Anecdotal reports by law enforcement officials, psychiatrists, and drug users themselves, as well as some surveys, link stimulants to aggression and unprovoked assaults. Users have committed murders and other violent acts while intoxicated with amphetamines. Nonetheless, the effects of stimulants on aggression and violence are complex and paradoxical. There are sharply differing opinions about the nature and extent of the problem (King and Ellinwood, 1997; Miczek and Tidey, 1989).

In a 1987 review of clinical observations and survey results, Miczek found differing representations of amphetamine effects (Miczek, 1987). Some surveys found that sizeable proportions of prison populations and juvenile delinquents commit crimes of violence while intoxicated by amphetamines, but other studies identified only rare cases and small percentages of juvenile delinquents and hostile persons who were amphetamine users. The reliability of these studies was unfortunately compromised by the lack of matched samples and reliance on verbal self-reports containing questionable information about dose and frequency of drug administration. It may be that intense violent acts are more prominent among chronic high-dose users, but no reports linked amphetamines to a high incidence of excessively violent behavior or other offensive social behavior (Miczek and Tidey, 1989).

Although well-controlled experimental studies of stimulant-associated aggression or violence in humans are scarce, the conclusions are similarly ambivalent. Early studies of cocaine or amphetamines did not focus on this aspect or note any increased aggression as a behavioral side effect. In fact, low doses (10 to 30 mg) of particular stimulants (i.e., Ritalin) have well-known and carefully studied beneficial effects on children 5 to 14 years old who are diagnosed with attention deficit/hyperactivity disorder (AD/HD) which manifests as aggressive, destructive, irritable, hyperactive behavior. Animal studies of mice, rats, squirrels, monkeys, and cats have examined aggression induced by isolation, pain, and brain stimulation in combination with amphetamines, with mixed results.

The most important determinants of aggression and defensive responses seem to be situation, species, prior experience with these behaviors, dosage, and chronicity of the stimulus. For example, a substantial increase in aggressive behavior can be evoked if amphetamines are administered to animals that are repeatedly confronting an intruder. Most importantly, there may be a biphasic dose effect on aggressive behavior in some animals: Aggression can be enhanced at low doses and also at higher doses, up to a point at which stereotypy and social withdrawal interfere (Miczek and Tidey, 1989; King and Ellinwood, 1997).

Other research more clearly confirms the effect of amphetamines on human aggression. A recent Japanese study ascertained that MA users scored higher on tests of verbal and physical aggressiveness and on impulsiveness than either alcoholic or normal control groups (Mukasa, 1990 [cited in Sowder and Beschner, 1993]). An earlier investigator found that participants in a task that enabled them to reward competitors with money or punish them with white noise increased their aggressiveness after 5 and 10 mg doses of amphetamines, whereas caffeine reduced the frequency of this aggressive behavior (Cherek et al., 1986 [cited in King and Ellinwood, 1997]).

Probably the most useful explanation of amphetamine effects on violence is offered by researchers who claim that stimulants do have a specific, but complex association with violent behavior. Chronic, moderate-to-high dose MA use, especially if the drug is injected or used by another rapid route of administration, often results in assaultive behavior and other forms of violence in the context of an interaction of behavioral and psychological effects (e.g., hyperactivity, agitation, emotional lability, and paranoid delusional thinking) combined with personality factors and social environment (King and Ellinwood, 1997). In other words, certain individuals who are regularly using high doses of amphetamine may be prone to intense violence, especially if experiencing paranoid delusions, but it is not known how frequently this occurs or what circumstances/personality characteristics promote this reaction (Miczek and Tidey, 1989).

Prevention of aggressive behavior

The Consensus Panel notes that the combination of low impulse control, paranoia, poor judgment, and grandiosity experienced by the chronic MA user, especially during a psychotic or prepsychotic episode, is a natural setup for violence. Similarly, the combination of a long-acting drug and a sustained high—because of MA's ready availability and low cost—results in a more severe/intense withdrawal reaction and accompanying susceptibility toward violence. The tweaker who is ready to crash after bingeing on ice does not need provocation to react aggressively, but confrontation increases the likelihood of a violent reaction.

Because drug-induced psychoses apparently increase the potential for violence in response to perceived persecution and paranoia, sound behavioral management techniques to prevent this negative and dangerous response are essential. The techniques listed in Figure 5-8 have been demonstrated to be useful and should be adopted by ER personnel as well as emergency medical technicians and police.

Co-Occurring Disorders Among People With Stimulant Use Disorders

Stimulant users have a surprising number of co- or preexisting disorders that can make differential diagnosis challenging or complicate treatment. Recently, investigators have become more interested in the implications of premorbid conditions as potential indicators of vulnerability to stimulant dependence. Majewska points out the need for more research to establish the epidemiological relationships between preexisting neurological deficits resulting from genetic, developmental, traumatic, or neurotoxic factors and vulnerability to drug addiction (Majewska, 1996). More specifically, preclinical studies and some surveys seem to indicate that neurological deficits associated with AD/HD, neuroanatomical abnormalities, lead poisoning, alcoholism, posttraumatic brain lesions, and posttraumatic stress disorder (PTSD) may be correlated with increased vulnerability to stimulant addiction. Another investigator (Bauer, 1996) lists another set of conditions or disorders that frequently co-occur with cocaine use disorders and notes that these correlates represent potential confounds to research regarding the sequelae of cocaine abuse and dependence as well as potential risk factors for developing those disorders. These include antisocial personality disorder, depression, other DSM Axis I disorders, polysubstance use, aggression, a family history of alcoholism or other substance use disorders, prescribed psychoactive medications, seizures, head injury, HIV/AIDS, and other major medical problems.

The following sections describe some of the most commonly identified premorbid and co-occurring disorders among stimulant users, with some comments on treatment precautions.

Polysubstance Use

Concomitant use of a variety of other licit and illicit psychoactive substances is a common correlate of stimulant use. These substances are frequently used to attenuate aversive symptoms experienced in the posteuphoric phase of use (Weis, 1997) or may be administered to prolong or counter particular effects of stimulant intoxication. Different combinations of

Figure 5-8
Recommended Approaches for Reducing the Risk of Violence

- **Keep the client in touch with reality by identifying yourself, using the client's name, and anticipating concerns.**
- Place the client in a quiet, subdued environment with only moderate stimuli. Ensure sufficient space so that the client does not feel confined. Have the door readily accessible to both the client and the interviewer, but do not let the client get between the interviewer and the door.
- Acknowledge agitation and potential for escalation into violence by reassuring the client that you are aware of his distress. Ask clear simple questions, tolerate repetitive replies, and remain nonconfrontational.
- Foster confidence by listening carefully, remaining nonjudgmental, and reinforcing any progress made.
- Reduce risk by removing objects from the room that could be used as weapons and discreetly ensuring that the client has no weapons.
- Be prepared to show force if necessary by having a backup plan for help and having chemical and physical restraints immediately available.
- Train all medical or emergency staff to work as a team in managing an aggressive, paranoid, and potentially violent client.

substances are used to titrate mood states or effects (CSAT, 1997).

Cocaine users tend to prefer alcohol, marijuana, or opiates. There is generally less alcohol use but more marijuana use among MA users than cocaine users (CSAT, 1997). Cigarette smoking is almost ubiquitous among stimulant users, usually to relieve perceived stress. *Speedballing*—simultaneous use of opioids and cocaine or other stimulants—is still prevalent in many places because the combination is perceived to smooth the effects of each drug. Some clients who are taking prescribed neuroleptics for psychiatric problems take stimulants to counteract the sedating properties of these antipsychotic medications (Weis, 1997).

Various reports indicate that 62 to 90 percent of cocaine users concurrently drink alcohol to prolong the high and attenuate unpleasant agitation and sleeplessness that emerge at the end of a binge (Gold, 1997; Gold and Miller, 1997). However, the combination of cocaine and alcohol appears to be particularly dangerous. Researchers have established that cocaethylene, an ethyl ester of benzoylecgonine, is formed in the liver when these two substances are used together and that this metabolite is particularly toxic to the liver. A substance user who combines cocaine and alcohol may experience more intense pleasure from the experience than using either substance alone, but is also exposed to the combined toxicities of cocaine and the even more potent cocaethylene (Gold, 1997; Cornish and O'Brien, 1996). Mendelson and colleagues found that a combination of ingested alcohol and injected MA increased users' perception of intoxication as well as cardiac responses, with potential for more serious cardiovascular consequences (Mendelson et al., 1995). Yamamura and colleagues found the combination aggravated both somatic and mental disorders (Yamamura et al., 1992). Because cocaethylene has a longer half-life (2 hours) than cocaine (38 to 60 minutes), the cumulative and additive effects found in the combination increase the incidence of lethal heart attacks and stroke (18 times higher risk of sudden death than with cocaine alone).

Cocaethylene appears to prolong the duration of cocaine-related increases in blood pressure and, in turn, to increase the likelihood of small vessel intercerebral infarcts. In addition, cocaethylene increases the risk of panic and anxiety attacks that chronic cocaine users experience, especially those that persist for some time. There is some indication that cocaethylene produces greater irritability and more persistent withdrawal complaints (Gold and Miller, 1997). The role of cocaethylene in evoking violence and intensifying agitation is also being investigated (Schrank, 1993).

Concomitant use of benzodiazepines and cocaine to blunt dysphoric effects is also common. This combination may enhance respiratory depression and prolong altered mental states, but decrease risk of seizures—especially if diazepam is taken before cocaine is used (Schrank, 1993).

The popularity of marijuana among stimulant users is explained by its pharmacologic properties. Because marijuana induces vasodilation of nasal mucosa, it attenuates the vasoconstriction of cocaine so that absorption is increased. Smoking marijuana before snorting cocaine decreases the time to peak euphoric effects, decreases dysphoric effects, and increases peak cocaine levels apparently by increasing bioavailability (Gold, 1997).

Psychiatric Disorders

It is believed that most stimulant users have concurrent psychiatric disorders. A 1991 survey of nearly 300 treatment-seeking cocaine users found that more than 70 percent had a lifetime history of psychiatric disorders such as alcoholism, major depression, bipolar disorder, anhedonia, anxiety, phobias, antisocial personality, and childhood AD/HD (Rounsaville and Carroll, 1991). At least four other earlier studies found similar comorbidity of cocaine with most of these same psychiatric diagnoses in addition to PTSD (Majewska, 1996). As many as half of surveyed cocaine users in treatment have lifetime diagnoses of depression; 20 to 25 percent have cyclic mood disorders; and sizeable percentages of these clients report borderline or antisocial personality, PTSD, or residual AD/HD (Gold, 1997). These psychiatric disorders are more common among stimulant users than in the general population (Weis, 1997).

Identified anxiety, phobias, AD/HD, and antisocial personality disorder typically precede cocaine dependence, whereas alcoholism, depression, and paranoia generally follow stimulant use. Although the symptoms of stimulant-induced psychosis closely mimic those of schizophrenia, and heavy use of cocaine/amphetamines may precipitate latent schizophrenia, the two disorders are not closely correlated (Majewska, 1996). Panic attacks are another correlate of cocaine use. Risk for this problem may increase because of sensitization to cocaine (Gold, 1997).

Differentiating comorbid psychiatric disorders from stimulant-related disorders can be challenging. Acute or chronic stimulant intoxication can elicit symptoms of anxiety that are indistinguishable from phobias, obsessive compulsiveness, panic, and generalized anxiety. The parallels between symptoms of stimulant-induced psychosis and schizophrenia are well known. Withdrawal from stimulants can cause depression that is indistinguishable from major depression from other causes (Gold and Miller, 1997). It can take at least a month of abstinence from all stimulant use to differentiate stimulant-induced dysphoria, depression, paranoia, or anxiety from a true psychiatric disorder.

The prognosis for substance use disorders is worsened by the presence of other untreated psychiatric disorders (or substance use disorders). Clients with comorbid psychiatric and drug dependence disorders need to have both treated; the psychiatric problems usually improve with abstinence. Antidepressant and neuroleptic medications with low anticholinergic and sedative properties are preferred in order to avoid another addiction. Sedative–hypnotics and benzodiazepines must be used with caution in high-risk populations (Gold and Miller, 1997).

Medical Conditions

Any preexisting acute or chronic physical conditions are also likely to be complicated and exacerbated by the stress of stimulant intoxication and withdrawal. Particularly dangerous coexisting medical conditions include any history of seizures, coronary heart disease, cardiac or thyroid problems, hypertension, or respiratory and pulmonary disease. Hypertension, renal failure, and diabetes mellitus, which are risk factors for stroke, can be exacerbated if cocaine/crack is smoked (Cornish and O'Brien, 1996).

Clients who are already taking medications for other medical conditions may be at special risk if stimulants are mixed with, for example, antidepressants, medications for high blood pressure, or antipsychotics. The effects of such drug interactions may be difficult to predict.

Crack- or MA-using mothers may be identified during prenatal care or in the delivery room through pregnancy or delivery complications, positive urine toxicologies, or acknowledged histories of substance use. Newborns who were exposed to stimulants in utero may manifest neurobehavioral problems that are less obvious and dangerous than those seen in opiate- or alcohol-exposed counterparts. The symptoms of stimulant exposure in newborns are likely to be transient and not require direct intervention. However, the young babies are typically irritable, tremulous, lethargic, emotionally labile, and somnolent. They may have a pronounced startle reaction, CNS instability, and prolonged and inconsolable crying. A few have signs of vascular disruptions and, rarely, congenital malformations, particularly of the heart, gastrointestinal tract, or skeletal system. Risk of sudden infant death syndrome may be heightened slightly.

Management is primarily by close observation in a quiet nursery environment, gentle handling, careful attention to feeding habits, and promoting positive bonding with the mother. More information about assessing, diagnosing, and managing the stimulant-exposed neonate can be found in TIP 5, *Improving Treatment for Drug-Exposed Infants* (CSAT, 1993).

Traumatic Injury

Patients appearing in hospital emergency departments following mild to severe traumas may be stimulant users who have been involved in fights or accidents of various types. The incidence of broken hands after fighting seems to be particularly high among MA users. TIP 16, *Alcohol and Other Drug Screening of Hospitalized Trauma Patients* (CSAT, 1995b), provides relevant information regarding how to identify and manage trauma patients with acute or chronic substance use disorders, including stimulant abuse. Several widely used screening instruments that can help hospital personnel determine substance use status of conscious trauma patients are described, as are laboratory tests to determine substance use status of any individual with potential symptoms of substance use, abuse, or dependence.

Assessment and Diagnosis

Diagnosis can be based on established DSM-IV criteria for amphetamine or cocaine abuse/dependence and other listed composites (American Psychiatric Press, 1994). For treatment reimbursement, the diagnosis may also need to reflect criteria according to the *International Classification of Diseases* (American

Medical Association, 1997). Arriving at a diagnosis is simplified by having information available from a relevant and accurate client history, a urine toxicology screen or similar laboratory tests, and clinical observations of physical signs and mental status.

History

An appropriate substance use history should include the substance(s) and medications used during the last 30 days; the specific substance(s) or combinations typically used with the usual dose, frequency, and route of administration; the duration of use; and the time and amount of last use, as well as when the symptoms or complaints developed and how they have progressed. If the client has been bingeing, a brief description of this and previous episodes is helpful. In addition, the history should include information about any previous seizures, delirium tremens, heart and pulmonary problems, paranoid reactions (with or without delusions and hallucinations), and other serious medical and psychological conditions and psychiatric diagnoses, as well as all current medications the client is taking. Although people with stimulant use disorders are not as likely as those with other types of substance use disorders (i.e., alcoholics) to have a genetic component or familial history, information about other substance abuse or psychiatric problems in the family can be enlightening.

For most patients presenting in an ER, the substance and medical history will, of necessity, be brief and focus on the potential causes for the observed symptoms and complaints and any potential medical or psychological problems that are likely to complicate management and the patient's response. Stabilize the patient medically before trying to take a history and assess potential danger to self or others; beware of exaggeration or dismissal by the patient of his symptoms and condition; and use significant others, whenever possible, to validate his history. In situations where the patient is delirious, psychotic, or unable to respond, information from accompanying friends or significant others about the antecedents of the problem is particularly important. Sometimes, the substance history must await symptomatic management.

The history may be supplemented by a variety of screening instruments constructed to ascertain substance use disorders, although these are not notably reliable if used with acutely psychotic or intoxicated individuals. A number of these screening instruments are described in detail in TIP 16, *Alcohol and Other Drug Screening of Hospitalized Trauma Patients* (CSAT, 1995b).

Urine Toxicology

A urine screen or toxicology test may be used to identify which substances the client has used recently. This testing is vital to confirm clinicians' personal assessments and observations. Some ERs have bedside or patient-side urine immunoassay testing kits (dipstick tests) that can be used for a quick turnaround without waiting on more formal assays. These can be validated by additional laboratory studies that require 6 to 8 hours or longer for processing in a hospital setting.

The results of either dipstick or Enzyme Multiplied Immunoassay Technique (EMIT) tests are appropriate to use for medical purposes, but cannot be used for criminal prosecution because no chain of custody is established. Alternative techniques for determining substance use are analyses of hair, blood, sweat, or tissue samples. In general, however, urine has become the standard method of determining substance use in an individual, and tests are readily available in the medical setting where other types of testing are not. Urine screens are less expensive than drawing blood samples for testing or other alternatives. Both qualitative and quantitative urine assays are usually needed to verify use and time/amount taken. Repeated assays may be used to track elimination of stimulants from the system if large amounts have been detected.

Because no standard set of substances is tested in a urine substance screen, medical personnel should make certain that assays for suspected substances are included. Also, no toxicology screen can determine with certainty that any particular substance—or any substances at all—was ingested. The detection limitations may be too broad or the specific substance may have been completely metabolized before a urine specimen was collected. A positive report will not necessarily indicate when the substance was last used: Metabolites for some substances are detectable for days or weeks after last use, but take some time after substance administration to be detectable in urine (CSAT, 1995b).

Stimulants can be detected in urine for approximately 24 to 48 hours following use and, maximally, for 3 days after a single dose and 7 to 12 days following repeated high doses (American Psychiatric Press, 1994). Cocaine is excreted more rapidly and is more difficult to detect in urine samples than MA. However, an EMIT test can detect benzoylecgonine, an inactive cocaine metabolite, in urine for up to 72 hours after last ingestion (Weis, 1997). Benzoylecgonine has been found in urine as late as 22 days after last cocaine intoxication in three asymptomatic clients with substantial histories of cocaine use (Goldfrank and Hoffman, 1993). Many prescription and over-the-counter drugs (e.g., diet aids, cold remedies) contain phenylpropanolamine or ephedrine that may yield positive EMIT or RIA tests for amphetamines. A procedure that does not have cross-reactivity to phenylpropanolamine or ephedrine will be needed to confirm that amphetamine was consumed (Hawks and Chiang, 1986).

Physical Signs and Mental Status

Data acquired from monitoring vital signs (temperature, blood pressure, pulse rate, respiration rate) can be used to document physical indicators. In addition, observations of physical manifestations listed for acute or chronic users and from the withdrawal stage can be documented. Similarly, a variety of instruments exists to determine mental status, although observational data regarding psychological and mental status may be adequate.

Differential Diagnosis

In the diagnostic process, other disorders and conditions with similar or identical presentations must be considered to rule out or include them. As already noted, many stimulant users have coexisting mental illnesses such as bipolar disorders, borderline personality, and so on. Similarly, the cause of a heart attack or seizure must be determined for optimal continuing care and medical management.

Before a differential diagnosis of a coexisting psychiatric disorder (dual diagnosis) can be made, the client must be abstinent for some period of time, at least 3 to 4 weeks. The syndrome and symptoms presented can be treated meanwhile, and a diagnosis of psychotic disorder, not otherwise specified (NOS), can be given. More information regarding the diagnostic process for clients with symptoms that indicate coexisting substance use and mood disorders can be found in TIP 9, *Assessment and Treatment of Patients With Coexisting Mental Illness and Alcohol and Other Drug Abuse* (CSAT, 1994a).

New forms of brain imaging techniques offer a promising approach for making a differential diagnosis if current research determines that these techniques are useful for distinguishing among drug-induced and other forms of psychosis.

Developing Linkages Between Treatment Programs and Medical Facilities

Because the ER may be the stimulant user's first point of contact with the medical system and potential treatment, attention needs to be given to establishing and supporting a continuum of care in which appropriate linkages among all necessary services and programs for substance users are represented. Although the burden of developing and encouraging these linkages among treatment components cannot fall to hospital staff alone, and it would be unrealistic to expect this, cooperation and enlightened self-interest are encouraged. If not hooked up to the treatment system, cocaine and MA users are likely to return repeatedly to the ER and other parts of the hospital for care of more and more serious health and mental health problems. Stimulant abuse/dependence, as all substance use disorders, is a life-long, relapsing condition that requires ongoing management and support.

Hence, treatment programs should take primary responsibility for developing linkages with hospitals, using several approaches. The most exemplary approach—and that most likely to succeed—is to have a substance abuse treatment counselor or trained nurse/social worker visit the hospital and other medical facilities regularly in order to identify, screen, encourage, and follow up clients who have stimulant-related and other substance use problems and need access to the ongoing treatment continuum. A face-to-face visit by an outreach specialist is particularly effective in supporting the crisis-precipitated motivation to enter treatment, especially if the potential client is hospitalized for some length of time. Because a crisis creates an intervention opportunity, clients may be unusually receptive to considering lifestyle alternatives and the need for longer term treatment.

It also may be realistic for hospital staff to hand out a list of available treatment facilities for stimulant use and/or other substance use disorders that is developed and provided by the substance use disorder treatment staff. However, it is not very likely that clients in crisis will follow up the suggested referral, especially if they are in the early stages of crashing (and terribly sleepy) or paranoid.

Some educational literature might also be helpful—particularly regarding withdrawal symptoms, drug-induced psychoses, and medical complications—if the client or a significant other is willing to read it. Because it is imperative for doctors and other medical staff to know about the addiction process in order to understand clients they see everyday, cross-training in the field of substance use disorder treatment is vital for learning about and actively supporting the development and use of linkages and referral mechanisms. It is believed that at least one-fourth of those treated in hospitals has some type of substance use–related problem.

Motivation for change is often difficult to determine in the substance user. Health problems may, however, be the motivation to move the individual from contemplation to action (Prochaska et al., 1992). Health care personnel working with a patient hospitalized for an acute drug episode may capitalize on the fact that the situation was so acute from drug use that he had to be hospitalized.

Hospitals often deal with a population known as "frequent flyers," that is, persons with frequent, revolving admissions to hospital ERs or inpatient hospital beds because of medical or psychiatric complications resulting from their substance use. The financial burdens can be severe for the patient and, in the case of those lacking insurance, the hospital's costs of care may be unrecoverable. A collaborative arrangement between the hospital and a local treatment facility can allow for door-to-door drug treatment.

Obtaining Consent for Treatment

In obtaining the client's consent for treatment, gathering information from others about his history of substance use, making referrals for continuing care, or seeking reimbursement from insurance carriers, hospital staff must be familiar with the provisions of special Federal and State laws and regulations for protection of clients' confidentiality as set forth in 42 U.S.C. §290dd-2 (1992) and C.F.R. Part 2. Intoxicated or psychotic clients may have diminished capacity for providing informed consent to treatment. If consent is obtained, even temporarily, from a relative, this may be considered a "disclosure of identifying information" and subject to Federal guidelines. In referring a client from a hospital to another treatment program and making an appointment, staff are also making a disclosure and will ordinarily need a written consent form from the client containing specified information (see Figure 5-9).

Special exceptions, however, apply to information needed in a medical emergency that can be provided to medical personnel who need health- or treatment-related facts about a client in order to treat his life-threatening condition. However, the treatment program that is, for example, providing this information to a hospital before transferring the client for emergency care, must document specific data in his record regarding the nature of the emergency, what information was released, the name of the person making the disclosure, and the date and time. Additional information about consent, confidentiality, and other types of communications governed by Federal regulations is presented in TIP 19, *Detoxification From Alcohol and Other Drugs*, (CSAT, 1995d).

Figure 5-9

Client Consent Form: Required Items

- **Name or general description of the program(s) making the disclosure**
- Name or title of the individual or organization that will receive the disclosure
- Name of the client who is the subject of the disclosure
- Purpose of or need for the disclosure
- How much and what kind of information will be disclosed
- A statement that the client may revoke the consent at any time, except to the extent that the program has already acted in reliance on it
- Date, event, or condition on which the consent form expires, if not previously revoked
- Signature of the client (and, for minors in some States, her parent or legal guardian)
- Date on which the consent is signed

6 Treatment Issues for Special Groups and Settings

This chapter has two purposes: (1) to provide specific recommendations for treatment tailored to the special needs of each group and (2) to underscore the need for cultural competence in the treatment setting. The second point is crucial to success in treatment for a variety of clients. The Consensus Panel feels strongly that cultural competence in the treatment setting extends beyond racial/ethnic sensitivity to understanding the mores of groups bound together by gender, age, geography, sexual preferences, criminal activity, substance use, and medical and mental illnesses. In addition, treatment providers need to understand the "culture" of their own organization, and determine how it may or may not be welcoming to members of other cultures.

This chapter discusses treatment issues specific to the following groups:

- Intravenous drug users
- Gay men
- Methadone maintenance clients
- Individuals with co-occurring mental disorders
- Medically ill clients (e.g., HIV, TB)
- Criminal justice clients
- Racial/ethnic minorities
- Rural populations
- Women
- Adolescents

Again, treatment for stimulant use disorders for members of any of these groups must occur against the backdrop of a solid understanding of the needs of the one or multiple groups in which a client may claim membership.

Intravenous Drug Users

The injecting cocaine user (ICU), like other injecting drug users (IDUs), poses a major public health problem by transmitting HIV and hepatitis. This transmission can occur in several ways. First, ICUs may spread infections by sharing injecting equipment with other IDUs. Second, ICUs may share needles with other populations that have their own independent risk for HIV and hepatitis, such as homosexuals and bisexuals. Third, ICUs may transmit the disease to nonsubstance users through sexual contact.

Prevalence of Injecting Drugs In Stimulant Users

Stimulant and other substance users make up the estimated 1 to 1.5 million IDUs in the United States. Approximately 85 percent of these IDUs do not receive any drug treatment services on any given day (Lurie and Drucker, 1997). Clearly, IDUs who are not in treatment are at great risk of suffering the many consequences of continued drug injection (Metzger et al., 1993).

The proportion of these IDUs who are ICUs can be best estimated by the National AIDS Demonstration Research (NADR) program, sponsored by the National Institute on Drug Abuse (NIDA). This program has provided the most comprehensive profile of active IDUs not in substance use disorder treatment. In that program 13,475 active IDUs were assessed from 28 sites across the country. The primary injected drugs were heroin (28 percent), cocaine (21 percent), and a combination of heroin and cocaine (35 percent). These estimates appear consistent with some treatment samples. For example, 20 percent of treatment-seeking cocaine users in the Los Angeles area were found to have injected drugs in the preceding year (Khalsa et al., 1992). Moreover, 94 percent of these ICUs reported sharing needles with other users. Together, these data suggest that somewhere between 20 percent and 51 percent (cocaine alone and combined cocaine–heroin users) of IDUs are cocaine users and that these users share injecting equipment that is putting them at risk for HIV, hepatitis, and other diseases.

Pattern of Use and Resulting Consequences

One factor that may increase risk of infection among ICUs is their pattern of use. Cocaine is frequently used in intermittent cycles of repeated multiple uses known as binges. Indeed, this pattern of use has been confirmed in human laboratory studies (Ward et al., 1997). This pattern often leads to more frequent injections during a binge than are generally observed in heroin-dependent populations. Moreover, injecting cocaine users often share needles with more people then those who report injecting other drugs.

Indeed, this greater frequency of injection during a binge appears to have as its consequence a greater likelihood of HIV infection. The frequent use of cocaine during binges, along with greater likelihood of shared needles, has been demonstrated to cause twice the risk of HIV infection for ICUs than for other IDUs (Chaisson et al., 1989; Anthony et al., 1991), and 1.5 times the risk of crack smokers (Kral et al., 1998).

Reducing Injection Drug Use And Its Consequences

A variety of interventions has been used to reduce the consequences of injection drug use (for reviews see Des Jarlais and Friedman, 1996; Sorenson, 1991). These interventions are tailored to injection drug users (IDUs) in general, yet the results from this research are more applicable to ICUs. It is important to note that education alone may not be effective in preventing the consequences of injecting drugs, because studies have shown that such programs increase knowledge without changing behavior.

Evidence shows that multicomponent HIV prevention programs, which include instruction on bleach disinfection along with skills training, counseling, and HIV testing, reduce the risk of transmission over time (Institute of Medicine [IOM], 1995). However, these results have not been supported in other studies, which failed to prove the efficacy of bleach disinfection. This may be caused by either ineffective disinfection procedures or inconsistent use of effective ones. The 1995 Report on the Prevention of HIV Infection, sponsored by the Institute of Medicine, recommends that bleach disinfection, when performed according to the guidelines provided by the Centers for Disease Control and Prevention (CDC) and the Center for Substance Abuse Treatment (CSAT), is likely to prevent HIV infection for IDUs who share injecting equipment. The IOM report recommends that IDUs be trained in effective procedures and more research be conducted to identify the simplest effective disinfection procedures.

Expanding that view, Des Jarlais and Friedman stated that, as currently implemented, bleach disinfection should not be relied on by HIV prevention programs (Des Jarlais and Friedman, 1996). They suggest, however, that bleach disinfection is more effective than not and should be performed when equipment is shared.

Needle exchange programs have been implemented in a wide variety of circumstances throughout the world (Hurley et al., 1997). The vast majority of studies and reports on needle exchange procedures suggests that they reduce the risk of HIV and hepatitis, and neither lead to more injecting drug use nor create new IDUs (Hurley et al., 1997; IOM, 1995).

Some populations received no noticeable benefit from these programs, however. For example, a drop in HIV infection was not seen in Montreal. One possible reason for this is that Montreal's needle exchange program restricted the number of needles a client could obtain per visit—fewer needles than ICUs typically use in the course of their drug use. Consequently, clients turned to other needle sources, negating the effect of the program.

Although these data tend to support the use of needle exchange programs, no controlled trial has yet to be conducted. The IOM report recommends that communities that desire such programs be permitted to use them. It also recommends that needle exchange be implemented as part of a multicomponent treatment, and that additional research and evaluation be conducted to evaluate the effects of needle exchange programs.

Gay Men

Research has found that men who have sex with men (MSMs) and who abuse alcohol, stimulants, inhalants, and other noninjection street drugs are more likely than non–substance users to engage in unprotected sex and become infected with HIV (Paul et al., 1991, 1993, 1994). Sexual risk-taking among MSMs, like that among the general population, often occurs under the influence of substance use, particularly stimulants. Sexual risk-taking within the context of substance use is hypothesized to occur due to disinhibition effects, learned patterns (especially between stimulant use and certain high-risk sexual practices), low self-esteem, altered perception of risk, lack of assertiveness to negotiate safe practices, and perceived powerlessness (Paul et al., 1993).

Alternatively, sex networks and sexual mixing patterns (Renton et al., 1995) might better explain the higher risk of HIV infection related to substance abuse among MSMs. As suggested by Renton and colleagues, MSM substance users may form tightly defined groups characterized by higher HIV seroprevalence rates, higher sexual mixing, greater injection drug use, and more trading sex for money, food, and drugs. These factors, rather than the suggested links between substance use and high-risk sex, would therefore account for higher HIV risks among MSM substance users. Regardless of which hypothesis better explains the connection between substance abuse and HIV/AIDS among MSMs, HIV/AIDS clearly intertwines with substance abuse. Unfortunately, similar to IDUs, it is estimated that only 10 percent of all MSMs who abuse substances seek therapy at substance use disorder treatment centers. Because of the stigmatization of MSMs, HIV-infected MSM substance users either do not seek treatment at traditional substance use disorder treatment centers, or they remain "closeted" when they do attend treatment.

Outreach

Outreach strategies for HIV prevention may readily translate to substance use interventions. In addition, there tends to be a stigma against substance users in the gay community, and outreach workers must be prepared to help clients overcome the stigma in order to get into treatment.

Of concern in this population is that any injection drug use, and use of injected methamphetamine (MA) in particular, increases the risk of HIV and/or hepatitis transmission from needles as well as from drug-induced bad judgment, feelings of invulnerability, risky behaviors, and repetitive and prolonged sexual behavior. A study of MA-using gay men in Los Angeles found that 62.5 percent of all participants reported having anal sex without a condom, and 56.3 percent reported having sex with someone who had HIV (Frosch et al., 1996). For counselors and outreach workers, risk assessment, including use analysis focused on how sex fits into the use patterns, is critical. It is important in the assessment process to capture these patterns.

Education of counselors, as well as clients, regarding the particular effects of this class of drugs is extremely important. Recently, there has been some backsliding with regard to injection drug use and sexual behaviors because of the perception that the new AIDS drugs mean a positive diagnosis is no longer automatically a death sentence. Not only does this myth need to be countered, but information on the effects of long-term stimulant use could diminish the attractiveness of MA-enhanced sexual performance. Side effects of long-term use include diminished sexual desire and performance. With ice in particular, clients show decreased ability to achieve orgasm, briefer erection periods, and an increase in impotence. Finally, counselor education needs to ensure lack of bias and sensitivity to the sexual practices of gay men.

Methadone Maintenance Clients

Cocaine and heroin are sometimes used together in a practice commonly known as *speedballing*. Some clients claim that methadone lengthens and mellows the effects of cocaine, presumably attenuating the negative reinforcers associated with cocaine crash (Condelli et al., 1991). Some patients also use alcohol or benzodiazepines or both concurrently with cocaine and heroin to reduce these effects of the cocaine crash, often marked by anxiety, depression, fatigue, and jitteriness. Thus, just as heroin use can increase the likelihood of cocaine dependence, cocaine use can increase the risk of heroin dependence (Dunteman et al., 1992).

Although methadone is very rarely combined with MA, there are some issues in this population of which counselors should be aware: treatment difficulties, medical risks, cocaine use, counselor training, and need for enhanced services.

Epidemiology

Stimulant abuse rates among methadone clients vary by program but range from 40 to 60 percent in inner city populations, according to data on positive screens for cocaine. These data suggest that the routes to MA use are established, and further study may be required to determine the extent to which they are used. In addition, data show a very high number of sedative users, including users of benzodiazepines, among methadone clients.

Individuals With Co-Occurring Mental Disorders

Individuals with co-occurring mental disorders may be defined as those persons with coexisting stimulant use and/or other substance use disorders and any of the following disorders:

- Major depression
- Dysthymia
- Bipolar disorder
- Antisocial personality disorder
- Panic disorder
- Agoraphobia
- Social phobia
- Posttraumatic stress disorder (PTSD)
- Attention deficit/hyperactivity disorder (AD/HD)
- Schizophrenia

It is critical to be aware of the fact that a DSM-IV diagnosis of a mental disorder is different from the mere production of symptoms (American Psychiatric Press, 1994). For example, most stimulant users will enter treatment exhibiting symptoms of depression. Similarly, many MA users will exhibit psychotic symptoms that are quite common in schizophrenics. However, the symptoms of depression are not the same as the psychiatric illness of depression, nor are the psychotic symptoms evidence of schizophrenia. Many of the symptom clusters, commonly associated with specific psychiatric disorders (e.g., depression, anxiety, psychosis, bipolar mood fluctuations, antisocial behavior) are frequently seen during the use of stimulants or during the period of early abstinence.

In fact, many individuals will bring their psychiatric diagnosis into their substance use disorder treatment evaluation. Many stimulant users have sought psychiatric care for their stimulant problems before entering treatment (many individuals apparently feel it is preferable to have a diagnosis of depression or bipolar illness to substance dependence). Therefore, they will contend that they have a psychiatric disorder and require psychiatric care (i.e., medication, psychotherapy) rather than substance use disorder treatment.

It should not be assumed that because an individual has received a previous psychiatric diagnosis, or because she has symptoms typically associated with a psychiatric disorder, that she is necessarily a "dual diagnosis" client. The accurate diagnosis of psychiatric comorbidity among stimulant users requires considerable diagnostic sophistication. It is often necessary to make a provisional diagnosis, which is modified after additional data are collected. For many cocaine users, it is often necessary to have 1 to 2 weeks of cocaine abstinence; for MA users, it is often helpful to have 30 days of abstinence to make an accurate psychiatric diagnosis. Another important element in the diagnosis is to obtain a careful history regarding the historical relationship between the onset of psychiatric symptoms and the substance use history.

Dual diagnosis will often encompass three or more coexisting conditions. Clients who use stimulants may or may not have an underlying psychiatric disorder, and it is often impossible to discern the source of behaviors symptomatic of a psychiatric condition until the client is substance-free. In many cases, a month's abstinence will be required before an accurate psychiatric assessment can be completed. Although treatment of clients with a dual diagnosis can be complex, this population often hits bottom faster and therefore enters treatment more quickly, and often with more motivation, than do clients who use substances without the more serious underlying problems.

Specialized Treatment Interventions

Consensus Panel suggestions for treatment interventions for persons with a stimulant use disorder and a coexisting psychiatric disorder are discussed in the sections below.

Affective disorders

Symptoms of depression may occur as part of the use of or withdrawal from stimulants. Where possible, it is helpful to wait to treat depression until the client has begun recovery from a substance use disorder, and the Consensus Panel recommends waiting to use medication to treat depressive symptoms if at all feasible. However, if the client is suicidal, hospitalization is the recommended course of action.

Bipolar disorder

Clients with bipolar disorder may be treated in traditional treatment settings if they are well controlled on their medications for the bipolar condition. Use of stimulants can initiate a manic episode. Therefore, medication management is one of the most important issues in treating clients with bipolar disorder.

Antisocial personality disorder

The diagnosis of antisocial personality disorders (ASP) is directly affected by substance use behavior. For example, many behaviors associated with chronic MA use mimic ASP, including law breaking, aggressiveness, and poor impulse control. Both cocaine use and ASP are associated with violence.

Many clients with ASP are involved with the criminal justice system, so coordination between systems (substance use disorder treatment, mental health, physical health) is important. Generally, men are more likely than women to present with ASP, and men typically use substances at a higher rate. Therefore, programs that are geared toward male clients with ASP and criminal involvement are necessary in a substance use disorder treatment system.

Panic disorder

Cocaine use can induce panic disorders, which can act as a trigger for panic attacks even after a client is substance-free. Panic disorders seem to be common for both cocaine and MA users long after they have discontinued using the drug and are often associated with depressions secondary to cocaine withdrawal. Health care providers should exercise extreme care in prescribing benzodiazepines for this disorder, due to their high addiction potential. Cognitive–behavioral techniques to recognize and manage symptoms may be some clinically useful strategies.

Posttraumatic stress disorder

Often, women who use ice are at an increased risk of PTSD because of substance-related episodes of domestic violence, sexual assault, and incest.

Recommended treatment approaches include referring clients to sexual assault and incest support groups as quickly as possible. Group counseling should be available in a woman-only format and should include coaching on what to expect from dreams, fears, and sleep disruptions as a result of PTSD and withdrawal from ice. Information on practical tools to combat nightmares such as night lights, herb teas, relaxation techniques, as well as information on relapse triggers, will provide clients with reassurance and skills to get through this period.

Counselors need special training to work with individuals with a co-occurring substance use disorder and PTSD. Issues include relapse triggers, timing of addressing issues in group sessions, and the tools and social skills women need in order to facilitate a successful recovery.

AD/HD

The prevalence of AD/HD in the general population ranges between 3 and 9 percent of adults. In the population of adults with substance use disorders, the prevalence of AD/HD ranges between 1 and 5 percent. However, one study has found that in the population of persons with adult AD/HD 40 percent had co-occurring substance use disorders, generally involving marijuana or alcohol (Biederman et al., 1993, 1995). These substance use disorders were detected when the clients entered treatment for AD/HD.

It is critical to retrieve a clear longitudinal history of both substance use and symptoms of AD/HD prior to completing a diagnosis of co-occurring disorder. Side effects of cocaine use can mirror some symptoms of AD/HD, but they disappear when use ceases. Generally, persons with adult AD/HD had the disease when they were children, although it may not have been diagnosed as such. It is important to include an assessment of childhood symptoms when completing the client's history. The presence of AD/HD symptoms in childhood provides a reliability measure for the presence of the adult disease.

Schizophrenia

Estimates of the prevalence of substance use disorders in the schizophrenic population range from 30 to 50 percent. Much of this use stems from peer group-seeking behaviors. Using substances is normal, it's what the other people on the streets are doing, and substance use can give persons with schizophrenia the sense of well-being and fitting they otherwise lack.

It is important to treat substance use in clients with schizophrenia immediately in order to allow medication for the mental illness time to take effect. A person with schizophrenia who continues to use cocaine will become fully psychotic, and those who continue to use MA will develop a psychosis indistinguishable from that created by paranoid schizophrenia.

After a stabilization period, treatment for both conditions can occur simultaneously with slight modifications. Modified group counseling may be used to treat substance use disorders. However, groups must be smaller and more controlled than in traditional substance use disorder treatment, and confrontational settings should be avoided. To be effective, each group session should focus on a particular skill or topic.

Medically Ill Clients

This section refers to clients who are undergoing treatment for a substance use disorder and who have one or more co-occurring medical disorders. Good clinical practice suggests that, prior to initiation of treatment for a substance use disorder, each client should be given a thorough physical examination. Followup should include contact with the clients and other providers and review of treatment records from them. In addition, treatment providers should be trained in the following:

- How to detect changes in medical conditions
- How to differentiate behaviors related to substance use from behaviors related to a deteriorating medical condition
- How to decide whether or not a referral to a physician is in order

Finally, it is important to note that linkages between substance use disorder treatment providers and the physicians and specialists who treat the client can improve treatment outcomes for the client.

This section discusses some medical conditions that require particular attention on the part of the substance use disorder treatment provider: epidemiology, diabetes, and HIV/AIDS.

Epidemiology

Chapter 5 of this TIP discussed some of the common medical illnesses seen in substance-using populations. Because the stimulant-using population tends to be younger, the medical conditions are not generally as severe as observed in the population of substance users as a whole. For example, because most persons who use stimulants range in age from 25 to 35 years, they do not present with conditions common to middle and old age such as coronary artery disease. Generally, primary medical problems are dental, dermatological, ear, nose, and throat, otitis media, nutritional (very thin clients who either use cocaine, have AIDS, or both), asthma, neurological disorders, seizures, and residual problems from a stroke. Secondary conditions include blood borne diseases, HIV, hepatitis, and sexually transmitted diseases.

Stimulant users in particular present higher rates of thyroid problems, in the form of permanent hyperthyroidism or hypothyroidism. This may be attributed to impure MA, resulting in a chemical, tissue-specific reaction in the thyroid, but more research needs to be performed in this area. For a complete list of the general medical complications of stimulant use, please refer to Chapter 5.

Hepatitis

Persons who use stimulants are often at risk for hepatitis, which, due to the ease of global travel, is no longer restricted to Third World countries. The Centers for Disease Control and Prevention (CDC) estimates that 150,000 people in the United States are infected each year by hepatitis A alone. The CDC lists household or sexual contact, sharing of infected needles, and recent international travel as the major known risk factors for transmission of hepatitis A. Hepatitis B virus (HBV) is a more virulent form of the disease and is much more prevalent than HIV, with an estimated 1.2 million Americans currently chronic carriers of HBV. Hepatitis B may develop into a chronic disease (which means lasting more than 6 months) in up to 10 percent of the 200,000 newly infected people each year. If left untreated, the risk of developing cirrhosis (scarring of the liver) and liver cancer is increased in clients with chronic hepatitis B. Treatment programs for stimulant users should include a screening for hepatitis in each client's initial assessment.

HIV/AIDS

Another important initial screening for persons with stimulant use disorders is that for HIV. Clients may be reluctant to undergo an HIV test because they fear the results. However, being diagnosed negative can act as a powerful motivator for stimulant users to comply with treatment more fully, once they no longer fear that they will develop AIDS. Testing negative can also encourage clients to practice preventive measures in other areas of their lives. Testing positive, although discouraging to the client, is an important part of the screening that the treatment provider needs to be aware of. Once the HIV status of the client is known, treatment can be planned that will include a medical component in the substance use disorder recovery program.

However, some special issues remain. One of the critical aspects of providing substance use disorder treatment to persons with HIV or AIDS is the continuing education that providers need regarding the changing and complex array of medication regimens available to this population. In addition, providers should know that clients with HIV/AIDS, armed with these new medicines, are approaching life differently. Now that they know they will live longer, they may return to using substances.

An issue with stimulant use is the increased risk of contracting HIV through the high-risk sexual behavior facilitated by the drug. By and large, clients have grown more aware of the risk of transmission from needles, although this is an area that requires further study. Cocaine users have more opportunities for exposure to HIV than do other stimulant users because they require multiple injections to maintain a high and therefore may run out of needles more quickly and be tempted to share. Bartering sex for drugs is a more likely route to HIV infection than unclean needles for MA users, because MA requires fewer injections.

Treating clients with HIV/AIDS is another area in which program linkages become critical to successful treatment. It is helpful, where possible, to have staff and nurses skilled in each type of treatment situated to provide clients convenient access. Anecdotal data from providers indicate that clients tend to get lost on the way to a referral appointment, and co-locating providers, such as an obstetrician–gynecologist in a methadone clinic, helps to mitigate this problem. For more information on this topic, please refer to *Treatment for HIV-Infected Alcohol and Other Drug Abusers* (TIP 15) (CSAT, 1995a; revision in press).

Criminal Justice Clients

A significant amount of crime is committed by substance users, and it is important to carefully assess this population. Many persons in the criminal justice system commit crimes while inhibitions are reduced by substance use, and others steal in order to buy drugs. These types of clients form the target treatment population. Other persons in the criminal justice system simply sell drugs but do not use them, or use drugs but are not addicted to the particular drug they sell. Self-report data indicate that 72 percent of persons in the criminal justice system are substance-dependent. These clients are often extremely complex in terms of case management: One client may have simultaneous involvement in the criminal justice, substance use disorder treatment, and mental health systems. There is little research on the specific issues faced by criminal justice clients with stimulant use disorders.

For more information on this topic, please refer to TIP 12, *Combining Substance Abuse Treatment With Intermediate Sanctions for Adults in the Criminal Justice System* (CSAT, 1994c); TIP 17, *Planning for Alcohol and Other Drug Abuse Treatment for Adults in the Criminal Justice System* (CSAT, 1995c); TIP 21, *Combining Alcohol and Other Drug Abuse Treatment With Diversion for Juveniles in the Justice System* (CSAT, 1995e); TIP 23, *Treatment Drug Courts: Integrating Substance Abuse Treatment With Legal Case Processing* (CSAT, 1996); and TIP 29, *Continuity of Offender Treatment for Substance Use Disorders From Institution to the Community* (CSAT, 1998b).

Racial/Ethnic Considerations

One of the most important issues in developing treatment options for different racial/ethnic groups is to move beyond cultural sensitivity and into cultural competence. Cultural competence is not just an understanding of superficial ethic designations but instead a knowledge of regional and socioeconomic patterns.

Cultural competence is critical for working in the drug treatment field today, because providers need to be culturally sensitive when working with diverse populations. Progressively increasing levels of cultural capacity include:

- Cultural sensitivity
- Cultural competence
- Cultural proficiency

Cultural sensitivity involves a basic understanding and appreciation of sociocultural factors as these relate to the client's treatment needs and the choice of relevant treatment. Cultural competence involves a greater depth of understanding of the client's needs within the client's cultural context. It also involves greater skills and experiences that allow working with cultural nuances as presented by the client, and the capacity to interpret deeper meanings in the client's thoughts and behaviors. Cultural competence aids in making better treatment decisions because it is based on a more effective matching of the client's needs with the relevant treatment options.

Outreach

Outreach issues will be specific to the targeted ethnic population, but topics to consider for each include

- Location of treatment centers and community-based organizations
- Availability of drop-in centers
- Availability of nonconfrontational programs
- Programs that spend more time in the engagement phase prior to counseling
- Programs that offer individualized approaches, encourage the establishment of safe relationships, allow time for trust to develop, and assess the level of motivation for change
- Counselors trained to help clients identify their own ethnic issues
- Counselors matched to clients by cultural competence (not necessarily racial/ethnic background)

Many of the above considerations involve slowing down the course of treatment to accommodate racial/ethnic mores. Of concern to the Consensus Panel is the method of payment for this type of care. Managed care is reluctant to fund long-term treatment or treatments that cannot be reduced to billable units.

Rural Populations

According to 1990 census data, approximately 25 percent of the U.S. population lives in rural areas. Colorado, Idaho, Montana, New Mexico, North Dakota, Nevada, South Dakota, Utah, and Wyoming have been identified as "frontier States," with at least half of their counties possessing six or fewer persons per square mile. All of these are western States—in which MA use is high.

The National Household Survey, conducted by the Substance Abuse and Mental Health Services Administration (SAMHSA), revealed that large metropolitan areas and rural areas had similar rates for substance use among youths 12 to 17 years old (SAMHSA, 1998). What differed was the specific substances used, not the prevalence of substance usage.

Strict regulatory controls on lawfully manufactured MA limit its diversion from licit to illicit channels. The bulk of MA currently on the streets has been illegally manufactured in clandestine labs. Rural areas are at high risk for being targeted by drug manufacturers and dealers. A rural community offers secluded areas in which to produce illegal drugs and access to major transportation routes for distribution with minimal risk of discovery by law enforcement. Small towns tend to be on major highways and interstates, which facilitates transportation to other regions for sale and distribution. This is a risk factor that many rural and/or frontier communities are unprepared for because they often lack the treatment resources or the community infrastructure, such as police forces trained specifically in dealing with these issues. This can lead to swift and severe problems for ill-prepared communities.

Prevalence in Rural Areas

The number of MA treatment facility admissions is highest in the western States where they now surpass cocaine admissions. Primary MA admission rates are beginning to rise in some southern and midwestern States.

In the State of Nevada, (a frontier State), the client data system for 1997 admissions to publicly funded substance use disorder treatment centers reported that of those individuals seeking treatment for substance use disorders, 52 percent were seeking treatment for MA use.

Challenges, Limitations, and Barriers to Treatment Services Faced by Rural Areas

Rural areas face the challenge of providing access to services for clients who may live hundreds of miles away from the nearest treatment provider. An entire day might be spent traveling by car to a service provider, because most rural areas lack public transportation.

Rural areas usually have a fragmented service system and limited resources. The lack of medical and social services in rural areas prohibits any continuum of care or even referral for specialized care. In many rural communities there are no treatment services available. Often only one social service provider is available and is generally overwhelmed by the various needs of the community. Social service agencies in rural areas tend to be multiservice agencies out of necessity.

Rural communities are generally allocated minimal funding for treatment because funding is usually based on population, which results in the provision of minimal services. There are rarely any sources within the community from which to seek additional funding. The lack of adequate funding limits staffing, staff salaries (which contributes to a high staff turnover), and the ability to provide support services such as childcare or transportation in the areas where that support is needed most.

Confidentiality is rare in rural settings. There is no anonymity in a small rural community. Everyone knows everyone and observes each other's comings and goings. Most rural communities' lack of available and appropriate office space leaves treatment providers practicing in whatever space they can find—which often involves little privacy.

Continuing education for treatment providers is nonexistent in most rural areas. Consequently, treatment providers often lack the most current information in the field.

Strategies To Provide Services In Rural Areas

The following are various strategies that can help provide treatment services to rural populations:

- Providers can form a consortium between educators, counselors, and law enforcement officers. By forming linkages between various agencies, small communities can have equal access to treatment services. The consortium can then use local statistics on substance use–related mortality rates, arrests for driving under the influence, school survey data, crime rates, and so on, to evaluate community needs. If data cannot be compiled easily by community members, the State substance abuse treatment office can either provide that information or direct individuals where to find it.
- Different providers can share staff and cross-train professionals and paraprofessionals. If the welfare worker is knowledgeable about addictions, she may be alert to symptoms of substance abuse in the client who comes in for food stamps. Conversely, the addictions counselor can identify other needs and provide the proper case management.
- Treatment programs can provide basic training of medical personnel, community social workers, teachers, school counselors, law enforcement officers, judges, and child care providers within a community. This can go a long way toward stretching resources and raising community awareness of the issues.

- Programs can provide safe and substance-free living arrangements for clients while in treatment away from their home community. Funds for this can be especially designated, solicited through recovery community networking, or obtained through donations.
- Programs can provide treatment services that are flexible in scope and structure. For example, an intensive outpatient program might offer a 6-hour session on Saturday and Sunday instead of the more usual weekday sessions. Treatment providers can also practice flexibility by assigning homework, arranging phone check-in, having drug testing done by outlying clinics, using online communication, and holding weekend workshops or retreats instead of traditional weekly sessions.
- Nontraditional outreach sites can be used for treatment services. A treatment facility in a small rural community may not be realistic, but it may be feasible to employ a part-time person in a satellite office who travels to different satellite sites to provide outpatient services. Many rural States have mobile rural healthcare—a network of vans that take primary care medical services to rural and isolated communities. Substance use disorder treatment could become an additional service provided in this manner. Like the mobile approach, some rural States also contract treatment counselors to travel from one rural site to another during the course of a week and who work out of other community service providers' offices. Although many communities lack sufficient office space in which to set up a treatment center, nearly every community has a school and a church that can serve. There is generally a healthcare or government service office within a reasonable distance that may provide some space for treatment services on a limited basis.
- Programs can work to develop outreach efforts with sponsorship from State agencies. Such efforts can help foster a strong self-help network in rural communities. Every community has "recovered" and "recovering people." However, these individuals often have never considered organizing themselves into a mutual support network. Most chapters of organizations such as Alcoholics Anonymous, Narcotics Anonymous, and Rational Recovery have members who would gladly extend their help and experience in developing such a network.
- Continuing education videotapes are a possible resource for rural treatment providers. State substance abuse offices and urban treatment providers can provide videotapes for those who cannot attend or afford the ongoing training that is available in urban areas.

Women's Issues

Treating stimulant use disorders in women can involve a host of complex issues (including pregnancy, children, domestic abuse, and socioeconomic problems) that can impact diagnosis and treatment of stimulant use disorders. However, although women's use of stimulants has not been well studied, one recent study does point to a gender difference in women's response to cocaine (Lukas et al., 1996). In addition, in recent studies of MA use, the percentage of female MA users appears higher than with samples of cocaine or heroin users (Rawson et al., 1998a).

Epidemiology

Although MA use is traditionally associated with males, growing numbers of women are using this class of drug, for reasons ranging from a desire to lose weight to the wish to be a "superwoman" who must self-medicate to get through an overextended day. Data on women who were sexually abused as children suggest they use cocaine as adults in order to "feel better."

Outreach

There are a number of entry points in the system for women who might not present directly for treatment, including

- Pediatricians (mothers will take children to the doctor even when they will not go for their own problems)
- Child protective agencies
- Social service agencies
- Primary care providers
- Criminal justice system

Two types of barriers must often be addressed concerning outreach to women who use substances, including stimulants. First, internal barriers to seeking treatment for substance use disorders that include guilt, depression, fear of children being taken away, and fear of partners who are using or dealing drugs must be identified and mitigated. Second, external barriers to be examined include lack of accessibility to treatment programs, need for child care, or lack of community-based programs that prevent women from seeking treatment. Often, reducing just one barrier is enough to bring a woman into treatment. For example, treatment programs that provide child care may have higher participation levels than those that do not.

Treatment programs must focus on the physical health of the woman entering treatment. Anecdotal data suggest that women experience more rapid physical deterioration than men from MA and cocaine use, but there is no solid research base to support this observation. Generally, by the time women get into treatment they are sicker than their male counterparts. In addition, women who are not in the workplace may have used the drug for a longer period of time without detection than their working counterparts and will be in worse shape when they do enter treatment.

Treatment programs must also consider the type of stimulant used. Ice, because it is a drug that is often used in family or community settings, exacerbates all of the women's issues described in this section.

Treatment for women should involve a holistic approach, including consideration of the following:

- Relationships with family (after social functioning, issues of homelessness, social isolation)
- Treatment needs of children
- Domestic violence
- Parenting
- Life skills
- Education and vocational training
- Economic self sufficiency
- Reproductive health issues
- Education about long-term affects of using stimulants
- Mental health
- Dependency issues
- Self-esteem
- Independent living skills
- Nutrition
- Transportation
- Ethnic and cultural issues
- Day care and group counseling for children
- Gender specific groups/female group leaders
- Long-term effects of stimulants on reproductive health

One relapse issue to consider for female clients is the negative impact of long-term MA use on sexual performance—the drug may cause women to lose sexual desire and the ability to achieve orgasm. However, they may also resort to MA or cocaine use, if pressured for sex, in order to get through the experience. In addition, women who return to the community to live, but who are living in a dependent relationship, may need to trade sex for food and shelter. This pressure for sex can lead to a resumption of substance use.

Intensive outpatient programs, which at first glance may appear to be more accessible to women with children, in fact present their own barriers. Programs that require frequent onsite visits but do not provide child care onsite will not enhance compliance with the treatment regime.

Adolescents

Adolescents can present many issues of concern besides their stimulant use. The path to abuse and dependence for this group may start because of general substance and alcohol experimentation, negative peer group or gang exposure, attempts to self-medicate for undiagnosed mental health conditions, poor self image (e.g., the desire to lose weight), the need to fabricate confidence to facilitate criminal activities, or any combination of these and other issues confronting adolescents. Other contributing issues may include anxiety, depression, loneliness, coping with past sexual or physical victimization, homelessness, and teen pregnancy.

The adolescent population is comprised of three subgroups:

- Sixth through ninth graders (advanced children)
- Tenth graders through high school seniors
- Adolescents older than high school age

A common treatment principle across all subgroups is that, to adolescents, the concept of death is so remote as to render scare tactics useless in pursing lifelong sobriety. However, this population offers hope to treatment providers because adolescents are not yet entrenched in their illness, and if substances can be removed from their lives, they can move on and learn with the rest of their peer group. Although there are many problems inherent in treating this population, the rewards of success can be that much greater.

Epidemiology

Although national studies at this time do not show evidence of high usage rates of MA or cocaine, these substances warrant attention for at least two reasons. First, adolescents tend to be multiple substance users, so an increase in use rates reported for other substances and stimulants can be somewhat indicative of trends in use of MA, and second, adolescents tend to use substances that are convenient to obtain; therefore rates of use are likely to be higher in areas with ample availability.

Nationally, the 1996 Monitoring the Future Study revealed an increase in opiate and cocaine use among 8th, 10th, and 12th graders, and teens in particular doubled opiate use (NIDA, 1998b). Amphetamines are not high on the list of drugs, with 2 to 3 percent of adolescent respondents reporting use during the period surveyed.

In terms of regional use by adolescents, problem areas include the West Coast, Midwest, and Hawaii. In California, rates of MA use among adolescents are higher than marijuana use. In Hawaii, data from Adolescent Drug Abuse Diagnosis (ADAD) (treatment programs that receive State funding) show that younger people (11 to 12 years old) are coming into treatment for ice use, indicating an even earlier initiation of gateway drugs leading to ice. In Arizona, although there are no data on the prevalence of MA use among adolescents, the proliferation of labs in both the inner city and all over the region, including home-based labs, suggests higher availability and therefore higher use among youth in this State.

Cultural and socioeconomic factors may also predict use in the adolescent population. In Hawaii, use patterns among Hawaiian and Filipino youth are related to the higher use patterns of their cultural groups as a whole. By contrast, MA use is not yet a big issue among Native American adolescents. MA use by youth is higher in lower middle class and working class neighborhoods, but adolescents from all socioeconomic classes use MA.

Outreach

Outreach to the adolescent population must focus not only on identifying high-risk youth but must also on identifying the most effective methods to reach them. High-risk youth will often have low motivation, drop out of high school, and show early involvement in low-level criminal activity. Often they have a number of hard to treat psychiatric issues, such as conduct disorder, depression, and AD/HD.

Outreach workers should get to these youths as early as possible. Counselors, teachers, school administrators, and others who work with young adolescents need training to identify behaviors that, if left unaddressed, can contribute to later abuse of substances, including stimulants. Generally, the more problems observed in an adolescent, the more she will be at risk for substance and stimulant abuse and dependence.

Although data are not stimulant-specific, it is useful for persons who work with adolescents to know that very often there is a psychiatric disorder that precedes the substance use disorder. It is important to treat the psychiatric problems in order to prevent self-medication. In addition, this group of adolescents tends to be impulsive, which puts them at higher risk for using substances. Adolescents with depression and conduct disorders are more likely to get involved in substance use than are their counterparts who are not dealing with mental health issues.

Because the public substance use disorder treatment system is often adult-driven, it is hard to know where to refer adolescents for screening and assessment. Often they show up in the juvenile justice system. When adolescents are referred for a discipline problem, it is important to screen for coexisting or underlying mental health and substance use disorders. A number of good screening and assessment tools exist for this purpose—for a list of them, please refer to the upcoming revised TIP, *Screening and Assessing Adolescents for Substance Use Disorders.* Adults who can serve as referral sources for adolescent substance users include pediatricians, teachers, school nurses, and school psychologists. This mix of professionals is important in order to cast a wide net for detecting adolescent stimulant use disorders.

A cautionary note for counselors new to this population—adolescents often experiment with substances and other risk-taking behaviors without developing dependence. Therefore, it is a mistake to automatically label experimental substance use behavior as a problem or dependence. With this population in particular, it is important not to over identify or label adolescents as substance abusers. (More information on this topic will be available in the revised TIP, *Screening and Assessing Adolescents for Substance Use Disorders* [CSAT, 1999a].)

Outreach also includes prevention and early intervention efforts. Although prevention efforts should begin earlier, middle school children, 11 to 12 years old, form an ideal target for comprehensive prevention programs. One example includes life skills–training programs for truant youths (a high-risk group whether or not they are using substances). Prevention efforts that take into consideration the influences of culture and family are important to consider.

In Hawaii, the police department runs a prevention program for Hawaiian youths that is grounded in the base of the local culture.

Treatment settings and approaches for adolescents include

- School-based clinics
- Traditional outpatient services
- Long-term residential
- Separate schools
- Developmental models
- Multisystemic therapy
- Experimental therapeutic camps
- Experimental wilderness-based programs

Programs for adolescents should focus on cessation of use as well as developing the life and educational skills that clients have missed while using substances. Adolescents need more intensity in programming, such as partial hospitalization, or attending treatment after school 5 days per week. It is generally better to avoid mixing adults and youths.

Programs for adolescents should involve the family, even when treatment takes place outside the home. Assessment should determine whether or not the family itself is substance-free. It is also important that assessment and treatment programs for adolescents be developmentally based.

Other Considerations

The adolescent population demands heightened attention because of the impact of stimulant and other substance use on this important developmental bridge into adulthood. Stimulant use can impede physical, emotional, and mental development.

Adolescents who use ice often move quickly into psychosis. This leads to a problem with differential diagnosis in cases of potential schizophrenia. With ice use, it can be difficult to determine whether psychosis is the result of schizophrenia or whether it was induced by MA use.

Stimulant use, especially of ice, may be a cause of an early onset of anorexia. Adolescents often abuse stimulants both to lose weight and to cope with sexual inhibitions. Dramatic weight loss in this population is a warning sign for MA use. However, physical health issues, other than anorexia and dental problems, are not as prevalent in the adolescent population as they are in the population of adults who use stimulants.

Teenage pregnancy is another concern because of the association of substance use and early sexual activity, compounded by impaired judgment regarding birth control and prevention of sexually transmitted diseases.

A final note on the adolescent population concerns the lack of community resources generally available to this group. Community-based organizations must be tapped, especially for adolescents from minority groups. Prevention of substance use disorders should be a part of the curriculum for professional groups and continuing education for any community member who works with the adolescent population. (More information on this topic will be available in the upcoming, revised TIP, *Treatment of Adolescents With Substance Use Disorders* [CSAT, 1999b]).

Appendix A
Bibliography

Alberti, R.E., and Emmons, M.L. *Your Perfect Right: A Guide to Assertive Living.* San Luis Obsipo, CA: Impact Press, 1982.

Allcott, J.V., III; Barnhart, R.A.; and Mooney, L.A. Acute lead poisoning in two users of illicit methamphetamine. *Journal of the American Medical Association* 258:510–511, 1987.

Amass, L. Financing voucher programs for pregnant substance abusers through community donations [abstract]. In: Harris, L.S., ed. *Problems of Drug Dependence 1996: Proceedings of the 58th Annual Scientific Meeting of the College on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 174. DHHS Pub. No. (ADM) 97-4236. Rockville, MD: National Institute on Drug Abuse, 1997. p. 60.

American Medical Association. *International Classification of Diseases,* 9th revision clinical modification (ICD-9-CM). Chicago: American Medical Association, 1997.

American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders,* 4th ed. Washington, DC: American Psychiatric Association, 1994.

Anglin, D.; Spears, K.L.; and Hutson, H.R. Flunitrazepam and its involvement in date or acquaintance rape. *Academic Emergency Medicine* 4(4):323–326, 1997.

Anglin, M.D.; Kalechstein, A.; Magline, M.; Annon, J.; and Fiorentine, R. (UCLA Drug Abuse Research Center). *Epidemiology and Treatment of Methamphetamine Abuse in California: A Regional Report.* Rockville, MD: National Evaluation Data and Technical Assistance Center (NEDTAC), Center for Substance Abuse Treatment, April 1998.

Angrist, B. Amphetamine psychosis: Clinical variations of the syndrome. In: Cho, A.K., and Segal, D.S., eds. *Amphetamine and Its Analogs: Psychopharmacology, Toxicology, and Abuse.* San Diego, CA: Academic Press, 1994.

Anker, A.L., and Crowley, T.J. Use of contingency contracts in specialty clinics for cocaine abuse. In: Harris, L.S., ed. *Problems of Drug Dependence, 1981: Proceedings of the 43rd Annual Scientific Meeting, the Committee on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 41. NTIS Pub. No. 82-190760. Rockville, MD: National Institute on Drug Abuse, 1982. pp. 452–459.

Anthony, J.C.; Vlahov, D.; Nelson, K.E.; Cohn, S.; Astemborski, J.; and Solomon, L. New evidence on intravenous cocaine use and the risk of infection with human immunodeficiency virus type 1. *American Journal of Epidemiology* 134(10):1175–1189, 1991.

Azrin, N.H., and Besalel, V.A. *Job Club Counselor's Manual.* Baltimore, MD: University Park Press, 1980.

Azrin, N.H.; Naster, B.J.; and Jones, R. Reciprocity counseling: A rapid learning based procedure for marital counseling. *Behavioral Research and Therapy* 11:365–382, 1973.

Barry, C.T. "Treatment for methamphetamine abuse disorders." Presented at the Methamphetamine Interagency Task Force Federal Advisory Committee Meeting, Washington, DC, May 4–5, 1998.

Bauer, L.O. Psychomotor and electroencephalographic sequelae of cocaine dependence. In: Majewska, M.D., ed. *Neurotoxicity and Neuropathology Associated With Cocaine Abuse.* NIDA Research Monograph Series, Number 163. DHHS Pub. No. (ADM) 96-4019. Rockville, MD: National Institute on Drug Abuse, 1996. pp. 66–93.

Beebe, D.K., and Walley, E.J. Smokable methamphetamine (ICE): An old drug in a different form. *American Family Physician* 51:449–453, 1995.

Bell, D.S. The experimental reproduction of amphetamine psychosis. *Archives of General Psychiatry* 29(1):35–40, 1973.

Biederman, J.; Faraone, S.V.; Spencer, T.; Wilens, T.; Norman, D.; Lapey, K.A.; Mick, E.; Lehman, B.K.; and Doyle, A. Patterns of psychiatric comorbidity, cognition, and psychosocial functioning in adults with attention deficit hyperactivity disorder. *American Journal of Psychiatry* 150(12):1792–1798, 1993.

Biederman, J.; Wilens, T.; Mick, E.; Milberger, S.; Spencer, T.J.; and Faraone, S.V. Psychoactive substance use disorders in adults with attention deficit hyperactivity disorder (ADHD): Effects of ADHD and psychiatric comorbidity. *American Journal of Psychiatry* 152(11):1652–1658, 1995.

Bigelow, G.E.; Stitzer, M.L.; Griffiths, R.R.; and Liebson, I.A. Contingency management approaches to drug self-administration and drug abuse: Efficacy and limitations. *Addictive Behavior* 6:241–252, 1981.

Brady, K.T.; Lydiard, R.B.; Malcolm, R.; and Ballenger, J.C. Cocaine-induced psychosis. *Journal of Clinical Psychiatry* 52(12):509–512, 1991.

Brotman, A.; Witkie, S.M.; Gelenberg, A.J.; Falk, W.E.; Wojcik, J.; and Leahy, L. An open trial of maprotiline for the treatment of cocaine abuse: A pilot study. *Journal of Clinical Psychopharmacology* 8:125–127, 1988.

Budney, A.J., and Higgins, S.T. *A Community Reinforcement Plus Vouchers Approach: Treating Cocaine Addiction.* Therapy Manuals for Drug Addiction, Manual 2. DHHS Pub. No. (ADM) 98-4309. Rockville, MD: National Institute on Drug Abuse, 1998.

Burton, B.T. Heavy metal and organic contaminants associated with illicit methamphetamine production. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications.* NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 47–59.

Carroll, K.M. "Combining psychotherapy and pharmacotherapy treatments to improve drug abuse outcomes." Paper presented at Addictions '96: Treatments Across the Addictions, International Symposium, Hilton Head, SC, September 27–29, 1996.

Carroll, K.M.; Chang, G.; Behr, H.; Clinton, B.; and Kosten, T. Improving treatment outcome in pregnant, methadone-maintained women: Results from a randomized clinical trial. *American Journal on Addiction* 4:56–59, 1995a.

Carroll, K.M.; Nich, C.; and Rounsaville, B.J. Differential symptom reduction in depressed cocaine abusers treated with psychotherapy and pharmacotherapy. *Journal of Nervous and Mental Disease* 183(4):251–259, 1995b.

Carroll, K.M.; Power, M.E.; Bryant, K.; and Rounsaville, B.J. One year follow-up status of treatment seeking cocaine abusers. Psychopathology and dependence severity as predictors of outcome. *Journal of Nervous and Mental Disease* 181:71–79, 1993a.

Carroll, K.M.; Rounsaville, B.J.; and Gawin, F.H. A comparative trial of psychotherapies for ambulatory cocaine abusers: Relapse prevention and interpersonal psychotherapy. *American Journal of Drug and Alcohol Abuse* 17:229–247, 1991a.

Carroll, K.M.; Rounsaville, B.J.; Gordon, L.T.; Nich, C.; Jatlow, P.; Bisighini, R.M.; and Gawin, F.H. Psychotherapy and pharmacotherapy for ambulatory cocaine abusers. *Archives of General Psychiatry* 51:177–187, 1994a.

Carroll, K.M.; Rounsaville, B.J.; and Keller, D.S. Relapse prevention strategies for the treatment of cocaine abusers. *American Journal of Drug and Alcohol Abuse* 17:249–265, 1991b.

Carroll, K.M.; Rounsaville, B.J.; Nich C.; Gordon, L.; and Gawin F. Integrating psychotherapy and pharmacotherapy for cocaine dependence: Results from a randomized clinical trial. In: Onken, L.S.; Blaine, J.D.; and Boren, J.J., eds. *Integrating Behavioral Therapies With Medications in the Treatment of Drug Dependence.* NIDA Research Monograph Series, Number 150. DHHS Pub. No. (ADM) 95-3899. Rockville, MD: National Institute on Drug Abuse, 1995c. pp. 19–36.

Carroll, K.M.; Rounsaville, B.J.; Nich, C.; Gordon, L.T.; Wirtz, P.W.; and Gawin, F. One-year followup of psychotherapy and pharmacotherapy for cocaine dependence. *Archives of General Psychiatry* 51: 989–997, 1994b.

Carroll, K.; Ziedonis, D.; O'Malley, S.; McCance-Katz, E.; Gordon, L.; and Rounsaville, B. Pharmacologic interventions for alcohol- and cocaine-abusing individuals: A pilot study of disulfiram vs. naltrexone. *American Journal on Addictions* 2:77–79, 1993b.

Castro, F.G.; Barrington, E.H.; Sharp, E.V.; Dial, L.S.; Wang. B.; and Rawson, R. Behavioral and psychological profiles of cocaine users upon treatment entry: Ethnic comparisons. *Drugs and Society* 6:231–251, 1992.

Center for Substance Abuse Research, University of Maryland. Methamphetamine use in the western United States: An in-depth look. *Cesar Fax* 6(29), 1997.

Center for Substance Abuse Treatment. *State Methadone Treatment Guidelines.* Treatment Improvement Protocol (TIP) Series, No. 1. DHHS Pub. No. (SMA) 93-1991. Washington, DC: U.S. Government Printing Office, 1991.

Center for Substance Abuse Treatment. *Improving Treatment for Drug-Exposed Infants.* Treatment Improvement Protocol (TIP) Series, No. 5. DHHS Pub. No. (SMA) 95-3057 (reprint). Washington, DC: U.S. Government Printing Office, 1993.

Center for Substance Abuse Treatment. *Assessment and Treatment of Patients With Coexisting Mental Illness and Alcohol and Other Drug Abuse.* Treatment Improvement Protocol (TIP) Series, No. 9. DHHS Pub. No. (SMA) 94-2078. Washington, DC: U.S. Government Printing Office, 1994a.

Center for Substance Abuse Treatment. *Assessment and Treatment of Cocaine-Abusing Methadone-Maintained Patients.* Treatment Improvement Protocol (TIP) Series, No. 10. DHHS Pub. No. (SMA) 94-3003. Washington, DC: U.S. Government Printing Office, 1994b.

Center for Substance Abuse Treatment. *Combining Substance Abuse Treatment With Intermediate Sanctions for Adults in the Criminal Justice System.* Treatment Improvement Protocol (TIP) Series, No. 12. DHHS Pub. No. (SMA) 94-3004. Washington, DC: U.S. Government Printing Office, 1994c.

Center for Substance Abuse Treatment. *Treatment for HIV-Infected Alcohol and Other Drug Abusers.* Treatment Improvement Protocol (TIP) Series, No. 15. DHHS Pub. No. (SMA) 95-3038. Washington, DC: U.S. Government Printing Office, 1995a.

Center for Substance Abuse Treatment. *Alcohol and Other Drug Screening of Hospitalized Trauma Patients.* Treatment Improvement Protocol (TIP) Series, No. 16. DHHS Pub. No. (SMA) 95-3041. Washington, DC: U.S. Government Printing Office, 1995b.

Center for Substance Abuse Treatment. *Planning for Alcohol and Other Drug Abuse Treatment for Adults in the Criminal Justice System.* Treatment Improvement Protocol (TIP) Series, No. 17. DHHS Pub. No. (SMA) 95-3039. Washington, DC: U.S. Government Printing Office, 1995c.

Center for Substance Abuse Treatment. *Detoxification From Alcohol and Other Drugs.* Treatment Improvement Protocol (TIP) Series, No. 19. DHHS Pub. No. (SMA) 95-3046. Washington, DC: U.S. Government Printing Office, 1995d.

Center for Substance Abuse Treatment. *Combining Alcohol and Other Drug Abuse Treatment With Diversion for Juveniles in the Justice System.* Treatment Improvement Protocol (TIP) Series, No. 21. DHHS Pub. No. (SMA) 95-3051. Washington, DC: U.S. Government Printing Office, 1995e.

Center for Substance Abuse Treatment. *Treatment Drug Courts: Integrating Substance Abuse Treatment With Legal Case Processing.* Treatment Improvement Protocol (TIP) Series, No. 23. DHHS Pub. No. (SMA) 96-3113. Washington, DC: U.S. Government Printing Office, 1996.

Center for Substance Abuse Treatment. *Proceedings of the National Consensus Meeting on the Use, Abuse, and Sequelae of Abuse of Methamphetamine With Implications for Prevention, Treatment and Research.* DHHS Pub. No. (SMA) 96-8013. Rockville, MD: CSAT, Substance Abuse and Mental Health Services Administration, Department of Health and Human Services, 1997.

Center for Substance Abuse Treatment. *Substance Use Disorder Treatment for People With Physical and Cognitive Disabilities.* Treatment Improvement Protocol (TIP) Series, No. 29. DHHS Pub. No. (SMA) 98-3249. Washington, DC: U.S. Government Printing Office, 1998a.

Center for Substance Abuse Treatment. *Continuity of Offender Treatment for Substance Use Disorders From Institution to Community.* Treatment Improvement Protocol (TIP) Series, No. 30. DHHS Pub. No. (SMA) 98-3245. Washington, DC: U.S. Government Printing Office, 1998b.

Center for Substance Abuse Treatment. *Screening and Assessing Adolescents for Substance Use Disorders.* Treatment Improvement Protocol (TIP) Series, No. 31. DHHS Pub. No. 99-3282. Washington, DC: U.S. Government Printing Office, 1999a.

Center for Substance Abuse Treatment. *Treatment of Adolescents With Substance Use Disorders.* Treatment Improvement Protocol (TIP) Series, No. 32. DHHS Pub. No. 99-3283. Washington, DC: U.S. Government Printing Office, 1999b.

Chafetz, M.E.; Blaine, H.T.; and Hill, M.J. *Frontiers of Alcoholism.* New York: Science House, 1970.

Chaisson, R.E.; Bacchetti, P.; Osmond, D.; Brodie, B.; Sande, M.A.; and Moss, A.R. Cocaine use and HIV infection in intravenous drug users in San Francisco. *Journal of the American Medical Association* 261(4):561–565, 1989.

Chaney, E.F. Social skills training. In: Hester, R.K., and Miller, W.R., eds. *Handbook of Alcoholism Treatment Approaches.* Elmsford, NY: Pergamon Press, 1989. pp. 206–221.

Charness, M.E. Alcohol and the brain. *Alcohol Health and Research World* 14(2):85–89, 1990.

Cherek, D.R.; Kelly, T.H.; and Steinberg, J.L. Behavioral contingencies and d-amphetamine effects on human aggressive and non-aggressive responding. In: Harris, L.S., ed. *Problems of Drug Dependence, 1986: Proceedings of the 47th Annual Scientific Meeting of the Committee on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 67. DHHS Pub. No. (ADM) 86-1448. Rockville, MD: National Institute on Drug Abuse, 1986. pp. 184–189.

Childress, A.R. Cue reactivity and cue reactivity interventions in drug dependence. In: *Proceedings of the Symposia on Cocaine Addiction: Trends, Laboratory Research, Clinical Issues, and Treatment.* Monograph Series No. 1. The Chemical Dependency Research Working Group. New York State Office of Alcoholism and Substance Abuse Services, 1994. http://www.users.interport.net/~nama/mono1.htm#Helping Cocaine Patients [Accessed October 10, 1997]

Cho, A.K. Ice: A new dosage form of an old drug. *Science* 249: 631–634, 1990.

Christensen, J.D.; Kaufman, M.J.; Levin, J.M.; Mendelson, J.H.; Holman, B.L.; Cohen, B.M.; and Renshaw, P.F. Abnormal cerebral metabolism in polydrug abusers during early withdrawal using 31P MR spectroscopy. *Magnetic Resonance in Medicine* 35:658–663, 1996.

Coca-Cola Bottling of Shreveport, Inc., et al. vs. The Coca-Cola Company, a Delaware Corporation, 769 F. Supp. 671.

Cohen, S. Causes of the cocaine outbreak. In: Washton, A.M., and Gold, M.S., eds. *Cocaine: A Clinician's Handbook.* New York: Guilford Press, 1987. pp. 3–9.

Community Epidemiology Work Group, National Institutes of Health, National Institute on Drug Abuse. *Epidemiologic Trends in Drug Abuse.* Vol. 1. NIH Pub. No. 96-4126. Rockville, MD: National Institute on Drug Abuse, June 1996a.

Community Epidemiology Work Group, National Institutes of Health, National Institute on Drug Abuse. *Epidemiologic Trends in Drug Abuse.* Vol. 2. NIH Pub. No. 96-4127. Rockville, MD: National Institute on Drug Abuse, June 1996b.

Community Epidemiology Work Group, National Institutes of Health, National Institute on Drug Abuse. "Epidemiologic Trends in Drug Abuse: Advance Report." Presented at the annual meeting of the Community Epidemiology Work Group, Washington DC, June 24–27, 1997.

Condelli, W.S.; Fairbank, J.A.; Dennis, M.L.; and Rachal, J.V. Cocaine use by clients in methadone programs: Significance, scope, and behavioral interventions. *Journal of Substance Abuse Treatment* 8:203–212, 1991.

Cook, C.E. Pyrolytic characteristics, pharmacokinetics, and bioavailability of smoked heroin, cocaine, phencyclidine, and methamphetamine. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications.* NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 6–23.

Cook, C.E.; Jeffcoat, A.R.; Hill, J.M.; Pugh, D.E.; Patetta, P.K.; Sadler, B.M.; White, W.R.; and Perez-Reyes, M. Pharmacokinetics of methamphetamine self-administered to human subjects by smoking S-(+)-MA hydrochloride. *Drug Metabolism and Disposition* 21:717–23, 1993.

Cooper, J.; Bloom, F.; and Roth, R. *The Biochemical Basis of Neuropharmacology,* 6th ed. New York: Oxford University Press, 1991.

Cornish, J.W., and O'Brien, C.P. Crack cocaine abuse: An epidemic with many public health consequences. *Annual Review of Public Health* 17:259–273, 1996.

Corrigan, I.D. Substance abuse as a mediating factor in outcome from traumatic brain injury. *Archives of Physical Medicine and Rehabilitation* 76:302–309, 1995.

Costello, R.M. Alcoholism treatment and evaluation: In search of methods. II. Collation of two-year follow-up studies. *International Journal of the Addictions* 10:251–275, 1975.

Cregler, L.L., and Mark, H. Medical complications of cocaine abuse. *New England Journal of Medicine* 315:1495–1500, 1986.

Cunningham, J.K., and Thielemeir, M.A. *Trends and Regional Variations in Amphetamine-Related Emergency Admissions: California, 1984–1993.* Los Angeles, CA: Public Statistics Institute, 1995.

Cunningham, J.K., and Thielemeir, M.A. *Trends and Regional Variations in Amphetamine-Related Emergency Admissions: California, 1985–1994.* Los Angeles, CA: Public Statistics Institute, 1996.

Czuchry, M.; Dansereau, D.F.; Dees, S.M.; and Simpson, D.D. The use of node-link mapping in drug abuse counseling: The role of attentional factors. *Journal of Psychoactive Drugs* 27:161–166, 1995.

Dansereau, D.F.; Joe, G.W.; and Simpson, D.D. Attentional difficulties and the effectiveness of a visual representation strategy for counseling drug-addicted clients. *International Journal of the Addictions* 30:371–386, 1995.

Daras, M. Neurologic complications of cocaine. In: Majewska, M.D., ed. *Neurotoxicity and Neuropathology Associated With Cocaine Abuse.* NIDA Research Monograph Series, Number 163. DHHS Pub. No. (ADM) 96-4019. Rockville, MD: National Institute on Drug Abuse, 1996. pp. 43–65.

Des Jarlais, D.C., and Friedman, S.R. HIV epidemiology and interventions among injecting drug users. *International Journal of STDs and AIDS* 7 (suppl) 2:57–61, 1996.

Di Chiara, G. The role of dopamine in drug abuse viewed from the perspective of its role in motivation. *Drug and Alcohol Dependence* 38:95–137, 1995.

Di Chiara, G. Alcohol and dopamine. *Alcohol Health and Research World* 21(2):108–114, 1997.

Drug Enforcement Administration. *The Cocaine Threat to the United States.* Washington, DC: Department of Justice, Drug Enforcement Administration, 1995.

Drug Enforcement Administration. *Methamphetamine Situation in the United States: Drug Intelligence Report.* Washington, DC: Department of Justice, Drug Enforcement Administration, 1996.

Drug Enforcement Agency. *1996 National Narcotics Intelligence Consumers Committee Report: The Supply of Illicit Drugs to the United States.* Washington, DC: Department of Justice, Drug Enforcement Administration, 1997.

Dunteman, G.H.; Condelli, W.S.; and Fairbank, J.A. Predicting cocaine use among methadone patients: Analysis of findings from a national study. *Hospital and Community Psychiatry* 43:608–611, 1992.

Elk, R. Pregnant women and TB-exposed drug abusers: Reducing drug use and increasing treatment compliance. In: Higgins, S.T., and Silverman, K., eds. *Motivating Behavior Change Among Illicit-Drug Abusers: Research on Contingency Management Interventions.* Washington, DC: American Psychological Association, in press.

Ellinwood, E.H., Jr. The epidemiology of stimulant abuse. In: Josephson, E., and Carroll, E.E., eds. *Drug Use: Epidemiological and Sociological Approaches.* Washington, DC: Hemisphere, 1974.

Ellinwood, E.H., Jr. Emergency treatment of adverse reactions to CNS stimulants. *A Treatment Manual for Acute Drug Abuse Emergencies.* DHEW Pub. No. (ADM) 76-230. Rockville, MD: Alcohol, Drug Abuse and Mental Health Administration, 1975.

Ellinwood, E.H., Jr., and Lee, T.H. Dose- and time-dependent effects of stimulants. In: Asghar, K., and De Souza, E., eds. *Pharmacology and Toxicology of Amphetamine and Related Designer Drugs.* NIDA Research Monograph Series, Number 94. DHHS Pub. No. (ADM) 89-1640. Rockville, MD: National Institute on Drug Abuse, 1989. pp. 323–340.

Ellinwood, E.H., Jr.; Sudilovsky, A.; and Nelson, L.M. Evolving behavior in the clinical and experimental amphetamine (model) psychosis. *American Journal of Psychiatry* 130:1088–1093, 1973.

Fals-Stewart, W.; Birchler, G.R.; and O'Farrell, T.J. Behavioral couples therapy for male substance-abusing patients: Effects on relationship adjustment and drug-seeking behavior. *Journal of Consulting and Clinical Psychology* 64:959–972, 1996.

Festinger, D.S.; Lamb, R.J.; Kirby, K.C.; and Marlowe, D.B. The accelerated intake: A method for increasing initial attendance to outpatient cocaine treatment. *Journal of Applied Behavioral Analysis* 29:387–389, 1996.

Festinger, D.S.; Lamb, R.J.; Kountz, M.; Kirby, K.C.; and Marlowe, D.B. Pre-treatment dropout as a function of treatment delay and client variables. *Addictive Behaviors* 20:111–115, 1995.

Feucht, T.E., and Kyle, G.M. Methamphetamine use among adult arrestees: Findings from the Drug Use Forecasting (DUF) program. *National Institute of Justice Research in Brief* NCJ 161842, Nov. 1996.

Friedman, L.M.; Furberg, C.D.; and DeMets, D.L. *Fundamentals of Clinical Trials.* Boston: John Wright, PSG, 1983.

Frosch, D.; Shoptaw, S.; Huber, A.; Rawson, R.A.; and Ling, W. Sexual HIV risk among gay and bisexual male methamphetamine abusers. *Journal of Substance Abuse Treatment* 13(6):483–486, 1996.

Gawin, F.H., and Ellinwood, E.H., Jr. Cocaine and other stimulants—actions, abuse, and treatment. *New England Journal of Medicine* 318:1173–1182, 1988.

Gawin, F.H., and Khalsa-Denison, M.E. Is craving mood-driven or self-propelled? Sensitization and "street" stimulant addiction. In: Majewska, M.D., ed. *Neurotoxicity and Neuropathology Associated With Cocaine Abuse.* NIDA Research Monograph Series, Number 163. DHHS Pub. No. (ADM) 96-4019. Rockville, MD: National Institute on Drug Abuse, 1996. pp. 224–250.

Gawin, F.H., and Kleber, H.D. Cocaine abuse treatment. Open pilot trial with desipramine and lithium carbonate. *Archives of General Psychiatry* 41:903–909, 1984.

Gawin, F.H., and Kleber, H.D. Abstinence symptomatology and psychiatric diagnosis in cocaine abusers. *Archives of General Psychiatry* 43:107–113, 1986.

Gawin, F.H.; Kleber, H.D.; Byck, R.; Rounsaville, B.J.; Kosten, T.R.; Jatlow, P.I.; and Morgan, C. Desipramine facilitation of initial cocaine abstinence. *Archives of General Psychiatry* 46:117–121, 1989.

Gerstein, D.R.; Johnson, R.A.; Harwood, H.J.; Fountain, D.; Suter, N.; Malloy, K., eds. *Evaluating Recovery Services: The California Drug and Alcohol Treatment Assessment (CALDATA).* Washington, DC: National Academy Press, 1994.

Gold, M.S. Cocaine (and crack): Clinical aspects. In: Lowinson, J.H.; Ruiz, P.; Millman, R.B.; and Langrod, J.G., eds. *Substance Abuse: A Comprehensive Textbook,* 3rd ed. Baltimore: Williams & Wilkins, 1997.

Gold, M.S., and Miller, N.S. Cocaine (and crack): Neurobiology. In: Lowinson, J.H.; Ruiz, P.; Millman, R.B.; and Langrod, J.G., eds. *Substance Abuse: A Comprehensive Textbook,* 3rd ed. Baltimore: Williams & Wilkins, 1997.

Goldfrank, L.R., and Hoffman, R. S. The cardiovascular effects of cocaine—update 1992. In: Sorer, H., ed. *Acute Cocaine Intoxication: Current Methods of Treatment.* NIDA Research Monograph Series, Number 123. DHHS Pub. No. (ADM) 93-3498. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 70–109.

Golub, A.L., and Johnson, B.D. Crack's decline: Some surprises across U.S. cities. *National Institute of Justice Research in Brief,* July 1997.

Goode, E. *Drugs in American Society,* 4th ed. New York: McGraw-Hill, 1993.

Gorelick, D.A. Pharmacologic therapies for cocaine addiction. In: Miller, N.S., and Gold, M., eds. *Pharmacological Therapies for Alcohol and Drug Addictions.* New York: Marcel Dekker, 1994. pp. 143–157.

Gorski, T., and Miller, M. *Staying Sober: A Guide for Relapse Prevention.* Independence, MO: Independence Press, 1986.

Gottleib, A. *The Pleasures of Cocaine.* Berkeley, CA: And/or Press, 1976.

Grabowski, J.; Rhoades, H.; Elk, R.; Schmitz, J.; Davis, C.; Creston, D.; and Kirby, K. Fluoxetine is ineffective for treatment of cocaine dependence and concurrent opiate dependence: Two placebo controlled double-blind trials. *Journal of Clinical Psychopharmacology* 15:163–174, 1995.

Grant, B.F., and Harford, T.C. Concurrent and simultaneous use of alcohol with cocaine: Results of national survey. *Drug and Alcohol Dependence* 25:97–104, 1990.

Grant, S.; London, E.D.; Newlin, D.B.; Villemagne, V.L.; Liu, X.; Contoreggi, C.; Phillips, R.L.; Kimes, A.S.; and Margolin, A. Activation of memory circuits during cue-elicited cocaine craving. *Proceedings of the National Academy of Sciences* 93:12040–12045, 1995.

Griffith, J.D.; Cavanaugh, J.; Held, J.; and Oates, J.A. Dextroamphetamine: Evaluation of psychomimetic properties in man. *Archives of General Psychiatry* 26(2):97–100, 1972.

Hall, J.N.; Uchman, R.S.; and Dominguez, R. *Trends and Patterns of Methamphetamine Use in the United States.* Report prepared for the Department of Epidemiology and Statistical Analysis. NIDA Order No. 88MO31054801D. Miami, FL: Up Front Drug Information Center, 1988.

Hall, S.M.; Tunis, S.; Triffleman, E.; Banys, P.; Clark, H.W.; Tusel, D.; Stewart, P.; and Presti, D. Continuity of care and desipramine in primary cocaine abusers. *Journal of Nervous and Mental Disease* 182:570–575, 1994.

Hando, J., and Hall, W. Patterns of amphetamine use in Australia. In: Klee, H. ed. *Amphetamine Misuse: International Perspectives on Current Trends.* Amsterdam: Harwood Academic Publishers, 1997.

Havassy, B.E.; Wasserman, D.A.; and Hall, S.M. Relapse to cocaine use: Conceptual issues. In: Tims, F.M., and Leukefeld, C.G., eds. *Cocaine Treatment: Research and Clinical Perspectives.* NIDA Research Monograph Series, Number 135. DHHS Pub. No. (ADM) 93-3639. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 203–217.

Hawks, R.L., and Chiang, C.N. Examples of specific drug assays. In: Hawks, R.L., and Chiang, C.N., eds. *Urine Testing for Drugs of Abuse.* NIDA Research Monograph Series, Number 73. DHHS Pub. No. (ADM) 87-1481. Rockville, MD: National Institute on Drug Abuse, 1986. pp. 84–112.

Heischobar, B., and Miller, M.A. Methamphetamine abuse in California. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications.* NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 60–71.

Herning, R.I.; Guo, X.; Better, W.E.; Weinhold, L.L.; Lange, W.R.; Cadet, J.L.; and Gorelick, D.A. Neurophysiological signs in cocaine dependence: Increased EEG beta during withdrawal. *Biological Psychiatry* 41:1087–1094, 1997.

Higgins, S.T., and Budney, A.J. Treatment of cocaine dependence through the principles of behavior analysis and behavioral pharmacology. In: Onken, L.S.; Blaine, J.D.; and Boren, J.J., eds. *Behavioral Treatments for Drug Abuse and Dependence.* NIDA Research Monograph Series, Number 137. NTIS Pub. No. 94-169570. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 97–121.

Higgins, S.T., and Budney, A.J. From the initial clinic contact to aftercare: A brief review of effective strategies for retaining cocaine abusers in treatment. In: Onken, L.S.; Blaine, J.D.; and Boren, J.J., eds. *Beyond the Therapeutic Alliance: Keeping the Drug-Dependent Individual in Treatment.* NIDA Research Monograph Series, Number 165. DHHS Pub. No. (ADM) 97-4142. Rockville, MD: National Institute on Drug Abuse, 1997. pp. 25–43.

Higgins, S.T.; Budney, A.J.; Bickel, W.K.; Foerg, F.E.; and Badger, G.J. Alcohol dependence and simultaneous cocaine and alcohol use in cocaine-dependent patients. *Journal of Addictive Diseases* 13:177–189, 1994a.

Higgins, S.T.; Budney, A.J.; Bickel, W.K.; Foerg, F.E.; Donham, R.; and Badger, G.J. Incentives improve treatment retention and cocaine abstinence in ambulatory cocaine-dependent patients. *Archives of General Psychiatry* 51:568–576, 1994b

Higgins, S.T.; Budney, A.J.; Bickel, W.K.; Foerg, F.E.; Ogden, D.; and Badger, G.J. Outpatient behavioral treatment for cocaine dependence: One-year outcome. *Experimental and Clinical Psychopharmacology* 3:205–212, 1995.

Higgins, S.T.; Budney, A.J.; Bickel, W.K.; Hughes, J.R.; and Foerg, F. Disulfiram therapy in patients abusing cocaine and alcohol. *American Journal of Psychiatry* 150:675–676, 1993a.

Higgins, S.T.; Budney, A.J.; Bickel, W.K.; Hughes, J.R.; Foerg, F.; and Badger, G. Achieving cocaine abstinence with a behavioral approach. *American Journal of Psychiatry* 150:736–769, 1993b.

Higgins, S.T.; Delaney, D.D.; Budney, A.J.; Bickel, W.K.; Hughes, J.R.; Foerg, F.; and Fenwick, J.W. A behavioral approach to achieving initial cocaine abstinence. *American Journal of Psychiatry* 148:1218–1224, 1991.

Higgins, S.T.; Roll, J.M.; and Bickel, W.K. Alcohol pretreatment increases preference for cocaine over monetary reinforcement. *Psychopharmacology* 123:1–8, 1996.

Higgins, S.T., and Wong, C.J. Treating cocaine abuse: What does research tell us? In: Higgins, S.T., and Katz, J.L., eds. *Cocaine Abuse Research: Pharmacology, Behavior, and Clinician Application.* San Diego, CA: Academic Press, 1998.

Higgins, S.T.; Wong, C.J.; Budney, A.J.; English, K.T.; and Kennedy, M.H. Efficacy of incentives during outpatient behavioral treatment for cocaine dependence [abstract]. In: Harris, L.S., ed. *Problems of Drug Dependence 1996: Proceedings of the 58th Annual Scientific Meeting of the College on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 174. DHHS Pub. No. (ADM) 97-4236. Rockville, MD: National Institute on Drug Abuse, 1997. p. 75.

Hiller-Sturmhöfel, S. Signal transmission among nerve cells. *Alcohol Health and Research World* 19(2):128, 1995.

Hoff, A.L.; Riordan, H.; Morris, L.; Cestaro, V.; Wienke, M.; Alpert, R.; Wang, G.J.; and Volkow, N. Effects of crack cocaine on neurocognitive function. *Psychiatry Research* 60:167–176, 1996.

Huber, A.; Ling, W.; Shoptaw, S.; Gulati, V.; Brethen, P.; and Rawson, R. Integrating treatments for methamphetamine abuse: A psychosocial perspective. *Journal of Addictive Diseases* 16(4):41–50, 1997.

Hughes, P.H.; Coletti, S.D.; Neri, R.L.; Umann, C.F.; Stahl, S.; Sicilian, D.M.; and Anthony, J.C. Retaining cocaine-abusing women in a therapeutic community: The effect of a child live-in program. *American Journal of Public Health* 85:1149–1152, 1995.

Hurley, S.F.; Jolley, D.J.; Kaldor, J.M. Effectiveness of needle-exchange programmes for prevention of HIV infection. *Lancet* 349(9068):1797–1800, 1997.

Iguchi, M.Y.; Belding, M.A.; Morral, A.R.; Lamb, R.J.; and Husband, S.D. Reinforcing operants other than abstinence in drug abuse treatment: An effective alternative for reducing drug use. *Journal of Consulting and Clinical Psychology* 65:421–428, 1997.

Inaba, D.; Cohen, W.E.; and Holstein, M.E. *Uppers, Downers, All Arounders: Physical and Mental Effects of Psychoactive Drugs*, 2nd ed. Ashland, OR: CNS Productions, 1993.

Institute of Medicine. *Preventing HIV Transmission: The Role of Sterile Needles and Bleach*. Normand, J.; Vlahov, D.; and Moses, L.E., eds. Washington, DC: National Academy Press, 1995.

Irvine, G.D., and Chin, L. The environmental impact and adverse health effects of clandestine manufacture of methamphetamine. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications*. NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 33–46.

Iyo, M.; Nishio, M.; Itoh, T.; Fukuda, H.; Suzuki, K.; Yamasaki, T.; Fukui, S.; and Tateno, Y. Dopamine D2 and serotonin S2 receptors in susceptibility to methamphetamine psychosis detected by positron emission tomography. *Psychiatry Research* 50:217–228, 1993.

Jenkins, S.W.; Warfield, N.A.; Blaine, J.D.; Cornish, J.; Ling, W.; Rosen, M.I.; Urschel, H.; Wesson, D.; and Ziedonis, D. A pilot trial of gepirone vs. placebo in the treatment of cocaine dependency. *Psychopharmacology Bulletin* 28:21–26, 1992.

Jeri, F.R. Coca-paste smoking in some Latin American countries: A severe and unabated form of addiction. *Bulletin on Narcotics* 36(2):15–31, 1984.

Joe, G.W.; Dansereau, D.F.; and Simpson, D.D. Node-link mapping for counseling cocaine users in methadone treatment. *Journal of Substance Abuse* 6:393–406, 1994.

Johanson, C.E., and Fischman, M.W. The pharmacology of cocaine related to its abuse. *Pharmacological Reviews* 41:3–52, 1989.

Johnson, R.E.; Eisenberg, T.; Stitzer, M.L.; Strain, E.C.; Liebson, I.A.; and Bigelow, G.E. A placebo controlled clinical trial of buprenorphine as a treatment of opioid dependence. *Drug and Alcohol Dependence* 40:17–25, 1995.

Kang, S.Y.; Kleinman, P.H.; Woody, G.E.; Millman, R.B.; Todd, T.C.; Kemp, J.; and Lipton, D.S. Outcomes for cocaine abusers after once-a-week psychosocial therapy. *American Journal of Psychiatry* 148(5):630–635, 1991.

Khalsa, H.K.; Kowalewski, M.R.; Anglin, M.D.; and Wang, J. HIV-related risk behaviors among cocaine users. *AIDS Education and Prevention* 4(1):71-83, 1992.

Kidorf, M., and Stitzer, M.L. Contingent access to methadone maintenance treatment: Effects on cocaine use of mixed cocaine-opiate abusers. *Experimental and Clinical Psychopharmacology* 1:200–206, 1993.

King, G.R., and Ellinwood, E.H. Amphetamines and other stimulants. In: Lowinson, J.H.; Ruiz, P.; Millman, R.B.; and Langrod, J.G., eds. *Substance Abuse: A Comprehensive Textbook,* 3rd ed. Baltimore: Williams & Wilkins, 1997.

Kirn, W. Crank. *Time Magazine* 151(24):June 22, 1998.

Kissin, B.; Platz, A.; and Su, W.H. Selective factors in treatment choice and outcome in alcoholics. In: Mello, N.K., and Mendelson, J.H., eds. *Recent Advances in Studies of Alcoholism.* Washington, DC: U.S. Government Printing Office, 1971. pp. 781–802.

Kleber, H.D. Pharmacotherapy, current and potential, for the treatment of cocaine dependence. *Clinical Neuropharmacology* 18(suppl. 1):S96–S109, 1995.

Koob, G.F. Drugs of abuse: Anatomy, pharmacology and function of reward pathways. *Trends in Pharmacological Sciences* 13:177–184, 1992.

Koob, G.F., and LeMoal, M. Drug abuse: Hedonic homeostatic dysregulation. *Science* 278:52–58, 1997.

Kosten, T.R.; Rosen, M.I.; Schottenfeld, R.; and Ziedonis, D. Buprenorphine for cocaine and opiate dependence. *Psychopharmacology Bulletin* 28:15–19, 1992.

Kral, A.H.; Bluthenthal, R.N.; Booth, R.E.; and Watters, J.K. HIV seroprevalence among street-recruited injection drug and crack cocaine users in 16 US municipalities. *American Journal of Public Health* 88(1):108–113, 1998.

Kramer, J.C. Introduction to amphetamine abuse. *Journal of Psychedelic Drugs* 2:8–13, 1969.

Kranzler, H.R.; Bauer, L.O.; Hersh, D.; and Klinghoffer, V. Carbamazepine treatment of cocaine dependence: A placebo-controlled trial. *Drug and Alcohol Dependence* 38:203–211, 1995.

Kreek, M.J. Opiates, opioids and addiction. *Molecular Psychiatry* 1:232–254, 1996.

Lago, J.A., and Kosten, T.R. Stimulant withdrawal. *Addiction* 89:1477–1481, 1994.

Landry, M. An overview of cocaethylene, an alcohol-derived psychoactive cocaine metabolite. *Journal of Psychoactive Drugs* 24:273–276, 1992.

Landry, M. *Understanding Drugs of Abuse: The Processes of Addiction, Treatment, and Recovery.* Washington, DC: American Psychiatric Press, 1994.

Landry, M. *Overview of Addiction Treatment Effectiveness.* Rockville, MD: Substance Abuse and Mental Health Services Administration, Office of Applied Studies, 1995.

Landry, M. *Alcoholics Anonymous: Review of the Research.* Rockville, MD: Substance Abuse and Mental Health Services Administration, Office of Applied Studies, in press.

Leshner, A.I. Addiction is a brain disease, and it matters. *Science* 278:45–47, 1997.

Leshner, A.I. Treatment: Effects on the brain and body. In: Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *The National Methamphetamine Drug Conference: Conference Proceedings, May 28–30, 1997, Omaha, Nebraska.* Sponsored by ONDCP. Washington, DC: ONDCP, 1998. http://www.whitehousedrugpolicy.gov/drugfact/methconf/plenary2.html [Accessed June 23, 1998]

Lieberman, M.A.; Yalom, I.D.; and Miles, M.B. *Encounter Groups: First Facts.* New York: Basic Books, 1973.

Ling, W., and Shoptaw, S. Integration of research in pharmacotherapy for addictive disease: Where are we? Where are we going? *Journal of Addictive Diseases* 16:83–102, 1997.

London, E.D.; Cascella, N.G.; Wong, D.F.; Phillips, R.L.; Dannals, R.F.; Links, J.M.; Herning, R.; Grayson, R.; Jaffe, J.H.; and Wagner, H.N., Jr. Cocaine-induced reduction of glucose utilization in human brain. *Archives of General Psychiatry* 47(6):567–574, 1990.

Lukas, S.E.; Sholar, M.; Lundahl, L.H.; Lamas, X.; Kouri, E.; Wines, J.D.; Kragie, L.; and Mendelson, J.H. Sex differences in plasma cocaine levels and subjective effects after acute cocaine administration in human volunteers. *Psychopharmacology* 125(4):346–354, 1996.

Lurie, P., and Drucker, E. An opportunity lost: HIV infections associated with lack of a national needle-exchange programme in the USA. *Lancet* 349(9052):604–608, 1997.

Majewska, M.D. Cocaine addiction as a neurological disorder: Implications for treatment. In: Majewska, M.D., ed. *Neurotoxicity and Neuropathology Associated with Cocaine Abuse.* NIDA Research Monograph Series, Number 163. DHHS Pub. No. (ADM) 96-4019. Rockville, MD: National Institute on Drug Abuse, 1996. pp. 1–26.

Marlatt, G.A., and Gordon, J.R. *Relapse Prevention.* New York: Guilford Press, 1985.

McCrady, B.S.; Noel, N.E.; Abrams, D.B.; Stout, R.L.; Nelson, H.F.; and Hay, W.M. Comparative effectiveness of three types of spouse involvement in outpatient behavioral alcoholism treatment. *Journal of Studies on Alcohol* 47(6):459–467, 1986.

McLellan, A.T.; Arndt, I.O.; Metzger, D.S.; Woody, G.E.; and O'Brien, C.P. The effects of psychosocial services in substance abuse treatment. *Journal of the American Medical Association* 269:1953–1996, 1993.

Melega, W.P.; Raleigh, M.J.; Stout, D.B.; Huang, S.-C.; and Phelps, M.E. Ethological and 6-[^{18}F]fluor-L-DOPA-PET profiles of long-term vunerability to chronic amphetamine. *Behavioural Brain Research* 84:259–268, 1997a.

Melega, W.P.; Raleigh, M.J.; Stout, D.B.; Lacan, G.; Huang, S.C.; and Phelps, M.E. Recovery of striatal dopamine function after acute amphetamine- and methamphetamine-induced neurotoxicity in the vervet monkey. *Brain Research* 766(1–2):113–120, 1997b.

Mendelson, J.; Jones, R.T.; Upton, R.; and Jacob, P. III. Methamphetamine and ethanol interactions in humans. *Clinical Pharmacology and Therapeutics* 57(5):559–568, 1995.

Mendelson, J.H., and Mello, N.K. Management of cocaine abuse and dependence. *New England Journal of Medicine* 334:965–972, 1996.

Metzger, D.S.; Woody, G.E.; McLellan, A.T.; O'Brien, C.P.; Druley, P.; Navaline, H.; DePhilippis, D.; Stolley, P.; and Abrutyn, E. Human immunodeficiency virus seroconversion among intravenous drug users in- and out- of treatment: An 18-month prospective follow-up. *Journal of Acquired Immune Deficiency Syndrome* 6(9):1049–1056, 1993.

Meyers, R.J., and Smith, J.E. *Clinical Guide to Alcohol Treatment: The Community Reinforcement Approach.* New York: Guilford Press, 1995.

Miczek, K.A. Psychopharmacology of aggression. In: Iversen, L.L.; Snyder, S.H.; Iversen, S.D., eds. *Handbook of Psychopharmacology.* Vol. 19. New York: Plenum Press, 1987. pp. 183–328.

Miczek, K.A., and Tidey, J.W. Amphetamines: Aggressive and social behavior. In: Asghar, K., and De Souza, E., eds. *Pharmacology and Toxicology of Amphetamine and Related Designer Drugs.* NIDA Research Monograph Series, Number 94. DHHS Pub. No. (ADM) 89-1640. Rockville, MD: National Institute on Drug Abuse, 1989. pp. 68–100.

Milby, J.B.; Schumacher, J.E.; Raczynski, J.M.; Caldwell, E.; Engle, M.; Michael, M.; and Carr, J. Sufficient conditions for effective treatment of substance abusing homeless persons. *Drug and Alcohol Dependence* 43:23–38, 1996.

Miller, B.L.; Chiang, F.; McGill, L.; Sadow, T.; Goldberg, M.A.; and Mena, I. Cerebrovascular complications from cocaine: Possible long-term sequelae. In: Sorer, H., ed. *Acute Cocaine Intoxication: Current Methods of Treatment.* NIDA Research Monograph Series, Number 123. DHHS Pub. No. (ADM) 93-3498. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 129–146.

Miller, M., and Kozel, N.J. Introduction and overview. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications.* NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 1–5.

Miller, W.R. Motivation for treatment: A review with special emphasis on alcoholism. *Psychological Bulletin* 98:84–107, 1985.

Miller, W.R. Increasing motivation for change. In: Hester, R.K., and Miller, W.R., eds. *Handbook of Alcoholism Treatment Approaches: Effective Alternatives,* 2nd ed. Boston: Allyn & Bacon, 1995. pp. 89–104.

Miller, W.R., and Munoz, R.F. *How to Control Your Drinking,* rev. ed. Albuquerque, NM: University of New Mexico Press, 1982.

Miller, W.R., and Rollnick, S. *Motivational Interviewing: Preparing People to Change Addictive Behavior.* New York: Guilford Press, 1991.

Milmoe, S.; Rosenthal, R.; Blane, H.T.; Chafetz, M.E.; and Wolf, I. The doctor's voice: Postdictor of successful referral of alcoholic patients. *Journal of Abnormal Psychology* 48:590–84, 1967.

Monti, P.M.; Rohsenow, D.J.; Colby, S.M.; and Abrams, D.B. Coping and social skills training. In: Hester, R.K., and Miller, W.R., eds. *Handbook of Alcoholism Treatment Approaches: Effective Alternatives,* 2nd ed. Boston: Allyn & Bacon, 1995. pp. 221–241.

Mori, A.; Suzuki, H.; and Ishiyama, I. Three cases of acute methamphetamine intoxication: Analysis of optically active methamphetamine. *Nippon Hoigaku Zasshi* 46(4): 266–270, 1992.

Mueller, M.D., and Wyman, J.R. Study sheds new light on the state of drug abuse treatment nationwide. *NIDA Notes* 12(5):1–7, 1997.

Nash, J.M. Addicted. *Time* 149(18):69–76, 1997.

National Institute of Justice (NIJ), Office of Justice Programs, Department of Justice. *Crack, Powder Cocaine, and Heroin: Drug Purchase and Use Patterns in Six U.S. Cities.* ONDCP Research Report NCJ 167265. Washington, DC: National Institute of Justice, 1997a.

National Institute of Justice (NIJ), Office of Justice Programs, Department of Justice. *Drug Use Forecasting: 1996 Annual Report on Adult and Juvenile Arrestees.* Washington, DC: National Institute of Justice, 1997b.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). *Eighth Special Report to the U.S. Congress on Alcohol and Health From the Secretary of Health and Human Services, September 1993.* NIH Pub. No. 94-3699. Bethesda, MD: National Institutes of Health, 1994.

National Institute on Alcohol Abuse and Alcoholism (NIAAA). Neuroscience research and medications development. *Alcohol Alert* 33(PH 366), 1996.

National Institute on Drug Abuse (NIDA). *Methamphetamine Abuse.* NIDA Capsule Series (C-89-06). Revised September 1997. http://165.112.78.61/NIDA Capsules/NCMethamphetamine.html [Accessed May 4, 1998].

National Institute on Drug Abuse (NIDA). *Methamphetamine: Abuse and Addiction.* NIDA Research Report Series. NIH Pub. No. 98-4210. Rockville, MD: National Institute on Drug Abuse, April 1998a.

National Institute on Drug Abuse. *Monitoring the Future Study, High School and Youth Trends.* Rockville, MD: National Institute on Drug Abuse, 1998b. http://www.nida.nih.gov/NIDACapsules/NCMTFuture.html [Accessed May 5, 1998].

National Institute on Drug Abuse. NIDA expands research to meet challenge of methamphetamine abuse. *NIDA Notes,* Volume 13, 1998c. http://165.112.78.61/NIDA_Notes/NNVol13N1/DirRepVol13N1.html [Accessed December 9, 1998]

Nestler, E.J., and Aghajanian, G.K. Molecular and cellular basis of addiction. *Science* 278:58–63, 1997.

Nestor, T.A.; Tamamoto, W.I.; Kam, T.H.; and Schultz, T. Acute pulmonary oedema caused by crystalline methamphetamine. *Lancet* 2(8674):1277–1278, 1989.

Nunes, E.V.; McGrath, P.J.; Quitkin, F.M.; Ocepek-Welikson, K.; Stewart, J.W.; Koenig, T.; Wager, S.; and Klein, D.F. Imipramine treatment of cocaine abuse: Possible boundaries of efficacy. *Drug and Alcohol Dependence* 39:185–195, 1995.

Obert, J.L.; Rawson, R.A.; and Miotto, K. Substance abuse treatment for "hazardous users": An early intervention. *Journal of Psychoactive Drugs* 29(3):291–298, 1997.

O'Brien, C.P.; Childress, A.R.; McLellan, A.T.; and Ehrman, R. Developing treatments that address classical conditioning. In: Tims, F.M., and Leukefeld, C.G., eds. *Cocaine Treatment: Research and Clinical Perspectives.* NIDA Research Monograph Series, Number 135. DHHS Pub. No. (ADM) 93-3639. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 71–91.

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *Street Terms: Drugs and the Drug Trade*. ONDCP Drugs and Crime Data, Drugs and Crime Clearinghouse. ONDCP Research Report NCJ 151622. Washington, DC: Office of National Drug Control Policy, 1995.

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *National Methamphetamine Strategy*. Washington, DC: Office of National Drug Control Policy, April 1996.

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *Pulse Check: Methamphetamine Trends in Five Western States and Hawaii*. Washington, DC: Office of National Drug Control Policy, January 1997a. http://www.whitehousedrugpolicy.gov/drugfact/pulsechk/pcappa.html [Accessed January 19, 1998].

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *Pulse Check: National Trends in Drug Abuse*. Washington, DC: Office of National Drug Control Policy, Winter 1997b. http://www.whitehousedrugpolicy.gov/drugfact/pulsechk/pcindex.html [Accessed January 19, 1998].

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *Cocaine: Facts and Figures*. ONDCP Drugs and Crime Data, Drugs and Crime Clearinghouse. Pub. No. PK 24. Rockville, MD: Office of National Drug Control Policy, 1998a.

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *Methamphetamine: Facts and Figures*. ONDCP Drug Policy Information Clearinghouse. Pub. No. PK 29. Rockville, MD: Office of National Drug Control Policy, 1998b.

Office of National Drug Control Policy (ONDCP), Office of Justice Programs, Department of Justice. *The National Methamphetamine Drug Conference: Conference Proceedings, May 28–30, 1997, Omaha, Nebraska*. Washington, DC: Office of National Drug Control Policy, 1998c.

O'Malley, S.; Adamse, M.; Heaton, R.K.; and Gawin, F.H. Neuropsychological impairment in chronic cocaine abusers. *American Journal of Drug and Alcohol Abuse* 18:131–144, 1992.

Oro, A.S., and Dixon, S.D. Perinatal cocaine and methamphetamine exposure: Maternal and neonatal correlates. *Journal of Pediatrics* 111: 571–578, 1987.

Parker, M.W.; Winstead, D.K.; and Willi, F.J. Patient autonomy in alcohol rehabilitation: I. Literature Review. *International Journal of the Addictions* 14:1015–1022, 1979.

Paul, J.P.; Stall, R.; and Bloomfield, K.A. Gay and alcoholics. *Alcohol Health and Research World* 15(2):151–160, 1991.

Paul, J.P.; Stall, R.D.; Crosby, G.M.; Barrett, D.C.; and Midanik, L.T. Correlates of sexual risk-taking among gay male substance abusers. *Addiction* 89(8):971–983, 1994.

Paul, J.P.; Stall, R.; and Davis, F. Sexual risk for HIV transmission among gay/bisexual men in substance-abuse treatment. *AIDS Education and Prevention* 5(1):11–24, 1993.

Peat, M.A.; Warren, P.F.; and Gibb, J.W. Effects of single dose of methamphetamine and iprindole on the serotonergic and dopaminergic system of the rat brain. *Journal of Pharmacological and Experimental Therapy* 225:126–131, 1983.

Petersen, R.C. "Methamphetamine abuse research—a review." Unpublished manuscript. Rockville, MD: Center for Substance Abuse Treatment, 1996.

Pettinati, H. Diagnosing personality disorders in substance abusers. In: Harris, L.S., ed. *Problems of Drug Dependence, 1990: Proceedings of the 54th Annual Scientific Meeting of the Committee on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 105. DHHS Pub. No. (ADM) 91-1753. Rockville, MD: Institute on Drug Abuse, 1991. pp. 236–242.

Prochaska, J.O.; DiClemente, C.C.; and Norcross, J.C. In search of how people change: Applications to addictive behaviors. *American Psychologist* 47:1102–1114, 1992.

Rabin, S.M., and Little, B.B. Peripartum methamphetamine use in a large urban population. *Journal of Maternal and Fetal Medicine* 3:101–103, 1994.

Rawson, R.A. Relapse prevention programming in the treatment of opioid users. *Substance Abuse Bulletin* 2:1–5, 1986.

Rawson, R.A.; Huber, A.; Brethen, P.; and Ling, W. "Treatment response and treatment outcome of methamphetamine and cocaine users." Presented at American Society of Addiction Medicine, New Orleans, Louisiana, 1998a.

Rawson, R.A.; Huber, A.; Brethen, P.; Shoptaw, S.; and Ling, W. "Methamphetamine and cocaine: Comparison of reported effects and response to treatment." Presented at CPDD Satellite Conference on Methamphetamine, San Juan, Puerto Rico, 1996.

Rawson, R.A.; Obert, J.L.; and McCann, M.J. *The Matrix Intensive Outpatient Program Therapist Manual.* Los Angeles, CA: The Matrix Center, Inc., 1995.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; Castro, F.G.; and Ling, W. Cocaine abuse treatment: A review of current strategies. *Journal of Substance Abuse* 3(4): 457–491, 1991a.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; and Ling, W. *The Matrix Model of Outpatient Treatment for Alcohol Use and Dependency.* Beverly Hills, CA: The Matrix Center, Inc., 1991b.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; and Ling, W. Neurobehavioral treatment for cocaine dependency: A preliminary evaluation. In: Tims, F.M., and Leukefeld, C.G., eds. *Cocaine Treatment: Research and Clinical Perspectives.* NIDA Research Monograph Series, Number 135. DHHS Pub. No. (ADM) 93-3639. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 92–115.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; and Mann, A.J. Cocaine treatment outcome: Cocaine use following inpatient, outpatient, and no treatment. In: Harris, L.S., ed. *Problems of Drug Dependence, 1986: Proceedings of the 47th Annual Scientific Meeting, the Committee on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 67. DHHS Pub. No. (ADM) 86-1448. Rockville, MD: National Institute on Drug Abuse, 1986. pp. 271–277.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; Smith, D.P., and Ling, W. Neurobehavioral treatment for cocaine dependency. *Journal of Psychoactive Drugs* 22:159–171, 1990.

Rawson, R.A.; Obert, J.L.; McCann, M.J.; Smith, D.P.; and Scheffey, E.H. *The Neurobehavioral Treatment Manual.* Beverly Hills, CA: The Matrix Center, Inc., 1989.

Rawson, R.A.; Shoptaw, S.J.; Obert, J.L.; McCann, M.J.; Hasson, A.L.; Marinelli-Casey, P.J.; Brethen, P.R.; and Ling, W. An intensive outpatient approach for cocaine abuse treatment: The Matrix Model. *Journal of Substance Abuse Treatment* 12:117–127, 1995.

Rawson, R.A.; Washton, A.M.; and Shoptaw, S. "Psychoactive substance use and sexual behavior: A study and analysis." Presented at the American Society of Addiction Medicine, New Orleans, Louisiana, 1998b.

Renton, A.; Whitaker, L.; Ison, C.; Wadsworth, J.; and Harris, J.R. Estimating the sexual mixing patterns in the general population from those in people acquiring gonorrhea infection: Theoretical foundation and empirical findings. *Journal of Epidemiology and Community Health* 49:205–213, 1995.

Restak, R.R. *The Mind.* New York: Bantam Books, 1988.

Ricuarte, G.A.; Schuster, C.R.; and Seiden, L.S. Long-term effects of repeated methylamphetamine administration on dopamine and serotonin neurons in the rat brain: A regional study. *Brain Research* 193:153–163, 1980.

Robbins, T.W.; Cador, M.; Taylor, J.R.; and Everitt, B.J. Limbic striatal interactions in reward-related processes. *Neuroscience and Biobehavioral Reviews* 13:155–162, 1989.

Robinson, T.E., and Becker, J.B. Enduring changes in brain and behavior produced by chronic amphetamine administration: A review and evaluation of animal models of amphetamine psychosis. *Brain Research* 396:157–198, 1986.

Roll, J.M.; Higgins, S.T.; Steingard, S.; and McGinley, M. Use of monetary reinforcement to reduce the cigarette smoking of persons with schizophrenia: A feasibility study. *Experimental and Clinical Psychopharmacology* 6:157–161, 1998.

Rounsaville, B., and Caroll, K. Psychiatric disorders in treatment-entering cocaine abusers. In: Schober, S., and Schade, C., eds. *Epidemiology of Cocaine Use and Abuse.* NIDA Research Monograph Series, Number 110. DHHS Pub. No. (ADM) 91-1787. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 227–251.

Rowan-Szal, G.; Joe, G.W.; Chatham, L.R.; and Simpson, D.D. A simple reinforcement system for methadone clients in a community-based treatment program. *Journal of Substance Abuse Treatment* 11:217–223, 1994.

Rowbotham, M.C. Cocaine levels and elimination in inpatients and outpatients: Implications for emergency treatment of cocaine complications. In: Sorer, H., ed. *Acute Cocaine Intoxication: Current Methods of Treatment.* NIDA Research Monograph Series, Number 123. DHHS Pub. No. (ADM) 93-3498. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 147–155.

Samber, S. Methamphetamine spread creates need for more comprehensive approaches. *The NCADI Reporter* June 30, 1997. http://www.health.org/pressrel/itn/rep/19.htm [Accessed February 5, 1998].

Satel, S.L.; Southwick, S.M.; and Gawin, F.H. Clinical features of cocaine-induced paranoia. *American Journal of Psychiatry* 148(4):495–498, 1991.

Sato, M.; Chen, C.C.; Akiyama, K.; and Otsuki, S. Acute exacerbation of paranoid psychotic state after long-term abstinence in patients with previous methamphetamine psychosis. *Biological Psychiatry* 18:429–440, 1983.

Scandling, J., and Spital, A. Amphetamine-associated myoblobinuric renal failure. *Southern Medical Journal* 75: 237–240, 1982.

Schottenfeld, R.S.; Pakes, J.; Ziedonis, D.; and Kosten, T.R. Buprenorphine: Dose-related effects on cocaine and opioid use in cocaine-abusing opioid-dependent humans. *Biological Psychiatry* 34:66–74, 1993.

Schrank, K.S. Cocaine-related emergency department presentations. In: Sorer, H., ed. *Acute Cocaine Intoxication: Current Methods of Treatment.* NIDA Research Monograph Series, Number 123. DHHS Pub. No. (ADM) 93-3498. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 110–128.

Seiden, L.S.; Fischman, M.W.; and Schuster, C.R. Long-term methamphetamine induced changes in brain catecholamines in tolerant rhesus monkeys. *Drug and Alcohol Dependence* 1:215–219, 1976.

Selden, L.S. Neurotoxicity of methamphetamine: Mechanisms of action and issues related to aging. In: Miller, M.A., and Kozel, N.J., eds. *Methamphetamine Abuse: Epidemiologic Issues and Implications.* NIDA Research Monograph Series, Number 115. DHHS Pub. No. (ADM) 91-1836. Rockville, MD: National Institute on Drug Abuse, 1991. pp. 24–32.

Self, D., and Nestler, E. Molecular mechanisms of drug reinforcement and addiction. *Annual Review of Neuroscience* 18:463–495, 1995.

Shaner, A.; Roberts, L.J.; Eckman, T.A.; Tucker, D.E.; Tsuang, J.W.; Wilkens, J.N.; and Mintz, J. Monetary reinforcement of abstinence from cocaine among mentally ill patients with cocaine dependence. *Psychiatric Services* 48:807–810, 1997.

Shoptaw, S.; Frosch, D.; Rawson, R.A.; and Ling, W. Cocaine abuse counseling as HIV prevention. *AIDS Education and Prevention* 9(6):511–20, 1997.

Shoptaw, S.; Rawson, R.A.; McCabb, M.J.; and Obert, J.L. The matrix model of outpatient stimulant abuse treatment: Evidence of efficacy. *Journal of Addictive Diseases* 13(4): 129–141, 1994.

Siegel, R.K. Cocaine smoking. *Journal of Psychoactive Drugs* 14(4):271–359, 1982.

Siegel, R.K. Cocaine smoking: Nature and extent of coca paste and cocaine free-base abuse. In: Washton, A.M., and Gold, M.S., eds. *Cocaine: A Clinician's Handbook.* New York: Guilford Press, 1987. pp. 175–191.

Silverman, K.; Bigelow, G.E.; and Stitzer, M.L. Treatment of cocaine abuse in methadone maintenance patients. In: Higgins, S.T., and Katz, J.L., eds. *Cocaine Abuse: Behavior, Pharmacology, and Clinical Applications.* San Diego, CA: Academic Press, 1998.

Silverman, K.; Higgins, S.T.; Brooner, R.K.; Monyoya, I.D.; Cone, E.J.; Schuster, C.R.; and Preston, K.L. Sustained cocaine abstinence in methadone maintenance patients through voucher-based reinforcement therapy. *Archives of General Psychiatry* 53:409–415, 1996.

Sisson, R., and Azrin, N.H. The community reinforcement approach. In: Hester, R.K., and Miller, W.R., eds. *Handbook of Alcoholism Treatment Approaches: Effective Alternatives.* New York: Pergamon Press, 1989. pp. 242–258.

Snyder, S. *Drugs and the Brain.* New York: Scientific American Library, 1986.

Sorensen, J.L. Preventing HIV transmission in drug treatment programs: What works? *Journal of Addictive Diseases* 10(4):67–79, 1991.

Sowder, B., and Beschner, G., eds. "Methamphetamine: An illicit drug with high abuse potential." Unpublished report from NIDA contract no. 271-90-0002. Rockville, MD: T. Head and Company, Inc., 1993.

Spotts, J.V., and Spotts, C.A. *Use and Abuse of Amphetamine and Its Substitutes.* NIDA Research Issues, No. 25. Rockville, MD: National Institute on Drug Abuse, 1980.

Stanton, M.D., and Shadish, W.R. Outcome, attrition, and family–couples treatment for substance abuse: A meta-analysis and review of the controlled comparative studies. *Psychological Bulletin* 122:170–191, 1997.

Stapleton, J.M.; Morgan, M.J.; Phillips, R.L.; Wong, D.F.; Yung, B.C.; Shaya, E.K.; Dannals, R.F.; Liu, X.; Grayson, R.L.; and London, E.D. Cerebral glucose utilization in polysubstance abuse. *Neuropsychopharmacology* 13(1):21–31, 1995.

Stark, M.J. Dropping out of substance abuse treatment: A clinically oriented review. *Clinical Psychology Review* 12:93–116, 1992.

Stark, M.J.; Campbell, B.K.; and Brinkerhoff, C.V. "Hello, may we help you?" A study of attrition prevention at the time of the first phone contact with substance-abusing clients. *American Journal of Drug and Alcohol Abuse* 16:67–76, 1990.

Stitzer, M.L.; Bickel, W.K.; Bigelow, G.E.; and Liebson, I.A. Effects of methadone dose contingencies on urinalysis test results of poly-abusing methadone-maintenance patients. *Drug and Alcohol Dependence* 18:341–348, 1986.

Stitzer, M.L.; Bigelow, G.E.; and Gross, J. Behavioral treatment of drug abuse. In: Karsu, T.B., ed. *American Psychiatric Association Treatment Manual.* Washington, DC: American Psychiatric Association, 1989.

Stitzer, M.L., and Higgins, S.T. Behavioral treatment of drug and alcohol abuse. In: Bloom, F.E., and Kupfer, D.J., eds. *Psychopharmacology: The Fourth Generation of Progress.* New York: Raven Press, 1995. pp. 1807–1819.

Stitzer, M.L.; Iguchi, M.Y.; and Felch, L.J. Contingent take-home incentive: Effects on drug use of methadone maintenance patients. *Journal of Consulting and Clinical Psychology* 60:927–934, 1992.

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *National Household Survey on Drug Abuse.* Rockville, MD: Substance Abuse and Mental Health Services Administration, 1988.

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *National Household Survey on Drug Abuse.* DHHS Pub. No. (ADM) 89-1636. Rockville, MD: Substance Abuse and Mental Health Services Administration, 1989.

Substance Abuse and Mental Health Services Administration (SAMHSA), National Clearinghouse for Alcohol and Drug Information. *1993 Monitoring the Future Survey: Data Tables and Figures.* Washington, DC: U.S. Government Printing Office, 1993a.

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *National Household Survey on Drug Abuse.* DHHS Pub. No. (SMA) 93-2053. Rockville, MD: Substance Abuse and Mental Health Services Administration, 1993b.

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *National Household Survey on Drug Abuse.* DHHS Pub. No. (SMA) 97-3149. Rockville, MD: Substance Abuse and Mental Health Services Administration, 1996a.

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *Preliminary Estimates From the Drug Abuse Warning Network.* Advance Report No. 17, August, 1996b. http://www.health.org/pubs/dawn/index.htm [Accessed May 6, 1998].

Substance Abuse and Mental Health Services Administration (SAMHSA), Office of Applied Studies. *Preliminary Results From the 1997 National Household Survey on Drug Abuse.* Rockville, MD: Substance Abuse and Mental Health Services Administration, 1998. http://www.samhsa.gov/oas/nhsda/nhsda97/httoc.htm [Accessed December 7, 1998].

Tennant, F.S., and Rawson, R.A. Cocaine and amphetamine dependence treated with desipramine. In: Harris, L.S., ed. *Problems of Drug Dependence, 1982: Proceedings of the 44th Annual Scientific Meeting, the Committee on Problems of Drug Dependence.* NIDA Research Monograph Series, Number 43. NTIS Pub. No. 83-252692. Rockville, MD: National Institute on Drug Abuse, 1983. pp. 351–355.

Tinklenberg, J.R. The treatment of acute amphetamine psychosis. *A Treatment Manual for Acute Drug Abuse Emergencies.* DHEW Pub. No. (ADM) 76-230. Rockville, MD: Alcohol, Drug Abuse, and Mental Health Administration, 1975.

Trulson, M.E., and Trulson, V.M. Effects of chronic methamphetamine administration on tryptophan hydroxylase activity, [^{3}H]-serotonin synaptosomal uptake, and serotonin metabolism in rat brain following systemic tryptophan loading. *Neuropharmacology* 21:521–527, 1982a.

Trulson, M.E., and Trulson, V.M. Reduction in brain serotonin synthesis rate following chronic methamphetamine administration in rats. *European Journal of Pharmacology* 83:97–100, 1982b.

Tusel, D.J.; Piotrowski, N.A.; Sees, K.; Reilly, P.M.; Banys, P.; Meek, P.; and Hall, S.M. Contingency contracting for illicit drug use with opioid addicts in methadone treatment [abstract]. In: Harris, L.S., ed. *Problems of Drug Dependence 1994: Proceedings of the 56th Annual Scientific Meeting of the College on Drug Dependence.* NIDA Research Monograph Series, Number 153. DHHS Pub. No. (ADM) 95-3883. Rockville, MD: National Institute on Drug Abuse, 1995. p. 155.

Ulm, R.R.; Volpicelli, J.R.; and Volpicelli, L.A. Opiates and alcohol self-administration in animals. *Journal of Clinical Psychiatry* 56(suppl. 7):5–14, 1995.

van Gorp, W.G.; Altshuler, L.; Theberge, D.C.; Wilkins, J.; Dixon, W. Cognitive impairment in euthymic bipolar patients with and without prior alcohol dependence: A preliminary study. *Archives of General Psychiatry* 55(1):41–46, 1998.

Volkow, N.D.; Ding, Y.S.; Fowler, J.S.; and Wang, G.J. Cocaine addiction: Hypothesis derived from imaging studies with PET. *Journal of Addictive Diseases* 15:55–71, 1996.

Volkow, N.D.; Wang, G.J.; Fischman, M.W.; Foltin, R.W.; Fowler, J.S.; Abumrad, N.N.; Vitkum, S.; Logan, J.; Gatley, S.J.; Pappas, N.; Hitzemann, R.; and Shea, C.E. Relationship between subjective effects of cocaine and dopamine transporter occupancy. *Nature* 386:827–830, 1997a.

Volkow, N.D.; Wang, G.J.; Fowler, J.S.; Logan, J.; Gatley, S.J.; Hitzemann, R.; Chen, A.D.; Dewey, S.L.; and Pappas, N. Decreased striatal dopaminergic responsiveness in detoxified cocaine-dependent subjects. *Nature* 386:830–833, 1997b.

Wada, K., and Fukui, S. Relationship between years of methamphetamine use and symptoms of methamphetamine psychosis. *Japanese Journal of Alcohol and Drug Dependence* 25:143–158, 1990.

Wagner, G.C.; Seiden, L.S.; and Schuster, C.R. Methamphetamine-induced changes in brain catecholamines in rats and guinea pigs. *Drug and Alcohol Dependence* 4:435–438, 1979.

Wallace, B.C. Treating crack cocaine dependence: The critical role of relapse prevention. *Journal of Psychoactive Drugs* 42(2):213–221, 1992.

Ward, A.S.; Haney, M.; Fischman, M.W.; and Foltin, W. Binge cocaine self-administration in humans: Intravenous cocaine. *Psychopharmacology* 132:375–381, 1997.

Washton, A.M. Preventing relapse to cocaine. *Journal of Clinical Psychiatry* 49(suppl. 2):34–38, 1988.

Washton, A.M. *Cocaine Addiction: Treatment, Recovery, and Relapse Prevention.* New York: Norton, 1989.

Washton, A.M. *Cocaine Recovery Workbooks: Preventing Relapse.* Center City, MN: Hazelden Educational Materials, 1990a.

Washton, A.M. *Staying Off Cocaine: Cravings, Other Drugs, and Slips.* Center City, MN: Hazelden Foundation, 1990b.

Washton, A.M. *Step Zero: Getting to Recovery.* New York: HarperCollins, 1991.

Washton, A.M. Outpatient groups at different stages of substance abuse treatment: Preparation, initial abstinence, and relapse prevention. In: Brook, D., and Spitz, H., eds. *Group Psychotherapy of Substance Abuse.* Washington, DC: American Psychiatric Press, in press.

Washton, A.M., and Stone-Washton, N. Outpatient treatment of cocaine and crack addiction: A clinical perspective. In: Tims, F.M., and Leukefeld, C.G., eds. *Cocaine Treatment: Research and Clinical Perspectives.* NIDA Research Monograph Series, Number 135. DHHS Pub. No. (ADM) 93-3639. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 15–30.

Weddington, W.W.; Brown, B.S.; Haertzen, C.A.; Hess, J.M.; Mahaffey, J.R.; Kolar, A.F.; and Jaffe, J.H. Comparison of amantadine and desipramine combined with psychotherapy for treatment of cocaine dependence. *American Journal of Drug and Alcohol Abuse* 17:137–152, 1991.

Weinrieb, R., and O'Brien, C. Persistent cognitive deficits attributed to substance abuse. *Neurologic Complications of Drug and Alcohol Abuse (Neurologic Clinics)* 11(3):670–675, 1993.

Weis, D.A. "Stimulant use disorder (adult)." Unpublished report. Minneapolis, MN: Institute for Healthcare Quality/Health Risk Management, 1997.

Wells, E.A.; Peterson, P.L.; Gainey, R.R.; Hawkins, J.D.; and Catalano, R.F. Outpatient treatment for cocaine abuse: A controlled comparison of relapse prevention and twelve-step approaches. *American Journal of Drug and Alcohol Abuse* 20:1–17, 1994.

Wesson, D.R., and Smith, D.E. Cocaine: Its use for central nervous system stimulation including recreational and medical use. In: Petersen, R.C., and Stillman, R.C., eds. *Cocaine: 1977.* NIDA Research Monograph Series, Number 13. DHHS Pub. No. (ADM) 77-432. Rockville, MD: National Institute on Drug Abuse, 1977. pp. 137–152.

West, R., and Gossop, M. Overview: A comparison of withdrawal symptoms from difference drug classes. *Addiction* 89:1483–1489, 1994.

Wetli, C.V. The pathology of cocaine: Perspectives from the autopsy table. In: Sorer, H., ed. *Acute Cocaine Intoxication: Current Methods of Treatment.* NIDA Research Monograph Series, Number 123. DHHS Pub. No. (ADM) 93-3498. Rockville, MD: National Institute on Drug Abuse, 1993. pp. 172–182.

Wise, R.A. Neuroleptics and operant behavior: The anhedonia hypothesis. *Behavioral and Brain Science* 5:39–87, 1982.

Wise, R.A. Neurobiology of addiction. *Current Opinions in Neurobiology* 6:243–251, 1996.

Woody, G.E.; McLellan, A.T.; Luborsky, L.; and O'Brien, C.P. Psychotherapy in community methadone programs: A validation study. *American Journal of Psychiatry* 152:1302–1308, 1995.

Woolverton, W.L.; Ricuarte, G.A.; Forno, L.S.; and Seiden, L.S. Long-term effects of methamphetamine administration in rhesus monkeys. *Brain Research* 486:73–78, 1989.

Yamamura, T.; Hishida, S.; Hatake, K.; Taniguchi, T.; and Ouchi, H. Effects of methamphetamine and ethanol on learning and brain neurotransmitters in rats. *Pharmacological and Biochemical Behaviors* 42(3):389–400, 1992.

Appendix B
Client Worksheets

This appendix includes the following client worksheets:

1. Daily Schedule and Planner
2. Identifying External Cues and Triggers
3. Identifying Internal Triggers
4. Action Plan for Cues and Triggers
5. Action Plan for Avoidance Strategies
6. Feelings, Thoughts, and Behaviors
7. Permission To Relapse
8. Delayed Stimulant Withdrawal
9. What About Alcohol?
10. Action Plan for the Holidays
11. Evaluating Your Self-Efficacy Regarding Relapse
12. Increasing Your Self-Efficacy
13. Stress: Identifying Your Warning Signs
14. Anger: Identifying Your Warning Signs
15. Recovery-Related Stress Reducers
16. Learning To Solve Problems
17. Managing Your Anger
18. Selective Memory About Stimulant Use
19. Fantasies About Controlled Use
20. Those Ugly Reminders
21. Recreational and Leisure Activities
22. Examples of Recreational and Leisure Activities
23. Exercise and Recovery
24. Types of Exercise Activities
25. Nutritional Self-Assessment
26. The Food Guide Pyramid
27. My Nutrition Improvement Action Plan
28. Sample Behavioral Contract for Stimulant Abstinence
29. Components of a Functional Analysis
30. Preparing To Conduct a Functional Analysis: Identifying Your Triggers
31. The Functional Analysis Worksheet
32. Conducting a Functional Analysis of Your Stimulant Use
33. Functional Analysis: Important Points To Consider
34. Self-Management Planning
35. Self-Management Planning Worksheet
36. Relationship Happiness Scale
37. Daily Reminder To Be Nice
38. The Perfect Relationship
39. Positive Requests
40. Reciprocal Contract for Behavior Change
41. Improving Communications
42. Disagreements and Fights
43. Good and Poor Listening Skills
44. Recovery Self-Evaluation

Client Worksheet 1

Daily Schedule and Planner

Date: ______________

7:00 ______________

8:00 ______________

9:00 ______________

10:00 ______________

11:00 ______________

12:00 ______________

1:00 ______________

2:00 ______________

3:00 ______________

4:00 ______________

5:00 ______________

6:00 ______________

7:00 ______________

8:00 ______________

9:00 ______________

10:00 ______________

11:00 ______________

- Do you have a clinic visit today? What time is the appointment?
- When will you have breakfast, lunch, and dinner?
- Are you going to work or school today? When are those commitments?
- When is your 12-Step or other self-help meeting?
- Have you scheduled time for exercise?
- Have you scheduled time for recreation and leisure activities?

Client Worksheet 2

Identifying External Cues and Triggers

Stimulant cues are those things in your life that remind you of stimulant use and can trigger drug hunger. Below are lists of people, places, events, objects, and activities. Check those items around which or whom you have frequently used stimulants. Within each list, circle the item that you think is most strongly associated with your stimulant use.

People

- ☐ Drug dealers
- ☐ Employer
- ☐ Dates
- ☐ Friends
- ☐ Family members
- ☐ Neighbors
- ☐ Coworkers
- ☐ Spouse/lover
- ☐ ____________

Places

- ☐ Neighborhoods
- ☐ Hotels
- ☐ Certain freeway exit
- ☐ School
- ☐ Friend's home
- ☐ Worksite
- ☐ Bathrooms
- ☐ Downtown
- ☐ Bars and clubs
- ☐ Concerts
- ☐ Stash storage place
- ☐ ____________

Events

- ☐ Meeting new people
- ☐ Payday
- ☐ During work
- ☐ Before sex
- ☐ Anniversaries
- ☐ Group meetings
- ☐ Calls from creditors
- ☐ After work
- ☐ During sex
- ☐ Holidays
- ☐ Parties
- ☐ Before work
- ☐ Going out
- ☐ After sex
- ☐ ____________

Objects

- ☐ Paraphernalia
- ☐ Movies
- ☐ Credit cards
- ☐ Magazine
- ☐ Television
- ☐ ATM machines
- ☐ Pornography
- ☐ Cash
- ☐ ____________

Behaviors and Activities

- ☐ Listening to certain music
- ☐ Going out to dance or eat
- ☐ When hanging out with friends
- ☐ When driving
- ☐ After paying bills
- ☐ Before or during a date
- ☐ When home alone
- ☐ When dancing
- ☐ After an argument
- ☐ ____________________

Client Worksheet 3

Identifying Internal Triggers

Stimulant cues can include certain feelings and emotions that can trigger drug hunger. Below are lists of emotions, feelings, and circumstances. Check those items that, in the past, have been associated with your stimulant use. Within each list, circle the item that you think may be the internal trigger with which you may struggle the most.

"Negative" Feelings

- ☐ Feeling afraid
- ☐ Feeling angry
- ☐ Feeling ashamed
- ☐ Feeling anxious
- ☐ Feeling criticized
- ☐ Feeling depressed
- ☐ Feeling guilty
- ☐ Feeling hateful
- ☐ Feeling inadequate
- ☐ Feeling irritated
- ☐ Feeling jealous
- ☐ Feeling left out
- ☐ Feeling overconfident
- ☐ Feeling overwhelmed
- ☐ ______________

"Normal" Feelings

- ☐ Feeling bored
- ☐ Feeling embarrassed
- ☐ Feeling frustrated
- ☐ Feeling insecure
- ☐ Feeling lonely
- ☐ Feeling neglected
- ☐ Feeling nervous
- ☐ Feeling pressured
- ☐ Feeling relaxed
- ☐ Feeling sad
- ☐ Feeling tired
- ☐ ______________

"Positive" Feelings

- ☐ Feel like celebrating
- ☐ Feeling confident
- ☐ Feeling excited
- ☐ Feeling exhausted
- ☐ Feeling happy
- ☐ Feeling "normal"
- ☐ Feeling passionate
- ☐ Feeling sexually aroused
- ☐ Feeling strong
- ☐ ______________

- How do you feel immediately before using stimulants?
- Typically, how do you want to feel immediately before using stimulants?
- In the past few days, what were you feeling when you either used or wanted to use stimulants?

Client Worksheet 4

Action Plan for Cues and Triggers

Stimulant use becomes associated with certain people, places, activities, behaviors, and feelings. These act as reminders about your previous stimulant use. When you experience these reminders or cues and do nothing about them, they can develop into thoughts about using, feelings of craving, and possibly stimulant use. But the process can be interrupted.

Many external triggers can be avoided. All triggers can be defused. However, when you try to ignore the triggers, they can become overwhelming and lead to cravings. You should develop action plans so that you can avoid being exposed to cues and reminders, and if you are exposed, so that you can stop them from becoming triggers and cravings.

- Do you have the feeling that an upcoming event or situation will become a trigger for stimulant cravings? If so, avoid the event or situation if possible!

- Are you in a situation or at an event that is making you think about stimulants? Can you leave the situation? If you can, leave now!

- Are you stuck in a situation that is making you think about stimulants? If so, visualize a switch or lever in your mind. Imagine that you can move the lever from the "On" to the "Off" position and thereby turn off the drug thoughts. Have another picture ready to think about in place of the drug thoughts.

- Were you just in a situation that made you think about stimulant use or that provoked stimulant cravings? If so, take action now! Call your 12-Step sponsor, call your counselor or a sober friend, take a quick walk, do physical exercise, or engage in a relaxation exercise. Also, make plans to attend the next available 12-Step meeting.

- Imagine a situation during which you cannot leave but which makes you think about using stimulants. What specific steps would you take to stop having thoughts about stimulants? What would you do once you left the situation?

- Imagine that you just left a situation that made you think about using stimulants. You are now having thoughts about stimulant use and are feeling stimulant cravings. What specific steps would you take?

- In the previous example, imagine that you are at work and you have stimulant thoughts and cravings. What specific steps would you take?

- In the same example, imagine that it is 6:00 p.m. and you have stimulant thoughts and cravings. What specific steps would you take? What steps would you take if it happened at 11:00 p.m.?

Client Worksheet 5

Action Plan for Avoidance Strategies

Stopping your stimulant use is more than simply having the desire and determination to stop. Rather, it requires action and behavior. More specifically, it requires specific plans of action that can decrease the likelihood of encountering reminders of your stimulant use.

Because there are numerous reminders of stimulant use in your environment, it is essential that you take specific steps to avoid them. Some of these steps may seem unnecessary to you at first glance. But experience shows that decreasing the reminders of your stimulant use will reduce the chances of experiencing drug thoughts, triggers, and cravings.

Getting rid of drugs and paraphernalia

- What drugs do you have left in your possession? What paraphernalia did you use to prepare or use stimulants? What objects or things did you use when taking drugs (such as pornographic magazines and videotapes, phone numbers of prostitutes)?

- Do you have a non-using friend or family member who can help you to collect and throw these away? Can you arrange to do this today? Can you arrange to do it immediately prior to a counseling session or a 12-Step meeting?

Stopping contact with stimulant users

- With whom do you use stimulants? From whom do you obtain stimulants? Do you have their phone numbers written down? Do you have their phone numbers programmed on your phone? What steps can you take to break contact with dealers and users? Do you have family members or lovers who use stimulants? What are your plans to not use if they are still using?

Avoiding high-risk areas

- What neighborhoods, streets, houses, or other locations are especially associated with obtaining or using stimulants? Do you encounter these during your daily routine, such as going to work or to the treatment program? Are there any ways that you can avoid these high-risk areas? What is your specific plan to avoid them?

Being prepared for confrontations

- You will encounter people who will offer you drugs. What are some of the things that you can say to refuse these drugs and leave the situation?

Client Worksheet 6

Feelings, Thoughts, and Behaviors

When you use stimulants, things tend to get out of control. You spend more money than you mean to, you use more drugs than you intend, and you experience negative consequences because of your stimulant use. Because of this, you probably experienced embarrassment, shame, and guilt. These feelings are a normal part of the addiction process. To deal with these problems, you probably developed certain patterns that helped you cope and make it through another day.

- **Feelings:** Because you had to deal with intense feelings, such as shame or guilt, you may have shut down your emotions. You probably stopped talking about how you felt and stopped being concerned about how others felt.

- **Thoughts:** Because it was hard to face the fact that you were engaging in behaviors that you didn't really like, you may have developed ways of thinking that allowed you to believe that there was no problem or that the problem was someone else's. You may have denied to yourself and to others that there was a problem, you may have minimized the extent of the problem, you may have developed some type of justification for the situation, or you may have blamed someone else.

- **Behaviors:** Because it was hard to listen to other people tell you that you had a problem, you may have learned to walk away and stop the discussion, blown up in anger, or intimidated others so that they wouldn't confront you. Or you may have spent a lot of time alone so that no one would bother you.

Relapsive Feelings, Thoughts, and Behaviors

These types of feelings, thoughts, and behaviors are tools that you learned that helped you to survive while you were using stimulants. They need to be replaced by healthy tools that will help you to survive and grow when not using stimulants.

- What are some of the ways in which you shut down your feelings? What are the benefits and risks associated with this? What can you do to change this?

- What are some of the ways in which your thinking becomes distorted so that you don't have to face reality? What are the benefits and risks associated with this? What are some ways to help you avoid this?

- What are some of the behaviors that you engage in so that you can avoid difficult feelings or facing reality? What are the benefits and risks associated with this? What types of behaviors would be more healthy?

Client Worksheet 7

Permission To Relapse

Relapse is not an event. It is not simply using stimulants after a period of abstinence. Rather, like addiction and recovery, relapse is a process. Similarly, relapse does not suddenly appear out of nowhere. Like recovery, it generally begins with small steps that ultimately lead to full-blown relapse and a return to stimulant use.

One of these small steps that lead to relapse is making up reasons why starting to use stimulants again may be okay. For example, you might start daydreaming about certain circumstances and situations in which using stimulants would be permissible.

The problem is, fantasizing about situations can lead you to situations that are very high risk for relapse. All of a sudden, you "find yourself" in a dangerous situation and experience powerful cravings and urges. The good news is that by identifying examples in which you justify relapse, you can stop the chain of events and avoid relapse.

Self-medicating your feelings

When you feel depressed, angry, lonely, or scared, do you want to use stimulants? When you feel this way, how would you like to feel? Have you ever said something like:

- "I am depressed and feel like hell. What would it matter if I used or not?"
- "I feel lonely. Who would know if I used?"
- "I hate my job. I need to use to get through another day."

It just happened

Do you ever believe the fantasy that you have no choice or that unexpected things just happen to you? Do you ever hear yourself say, "It just happened. I didn't choose to do it?" Have you ever said something like:

- "An old friend called, we decided to get together, and he offered me some cocaine."
- "I was just cleaning the house and found some drugs that I had forgotten about."
- "It was offered to me. I didn't know what to say. I couldn't leave the bar."

Blame it on something else

Have you ever heard yourself blame another person or a situation for your own behavior? Have you ever used stimulants to get back at someone else? Have you ever said something like:

- "Well, if she didn't stay on my back all the time, I probably wouldn't use."
- "Why should I stay clean? He doesn't."
- "As long as she thinks that I am using, I might as well use."

Client Worksheet 8

Delayed Stimulant Withdrawal

Most people expect to experience several problems when they stop using stimulants. You may have been very sad or depressed, felt extremely tired and sleepy, had severe stimulant cravings, and found it very hard to concentrate. It is easy to recognize that these problems are directly related to abruptly stopping the use of stimulants. Most people call them withdrawal symptoms.

However, many people don't realize that some of these symptoms can reoccur a few months after last using stimulants. For example, after a few months, some people will feel sad or depressed, but generally not as depressed as during withdrawal. Some people will have a lack of energy and feel that they just don't care about things (apathy). Even though it may have been several months since you last used stimulants, you can experience a type of *delayed stimulant withdrawal*. These symptoms:

- Are a normal part of recovery
- Are part of the brain's healing process
- Are temporary
- Can be endured
- Can be lessened by participation in recovery efforts

Delayed withdrawal checklist

The items listed below can be part of a delayed stimulant withdrawal. How many of them are you experiencing?

☐ Sadness
☐ No energy
☐ Alcohol craving
☐ Poor memory
☐ Stopping exercise
☐ Being alone
☐ Feeling lonely
☐ Anxiety
☐ Mood swings
☐ Alcohol use
☐ Feeling hopeless
☐ Feeling uneasy
☐ Fuzzy thinking
☐ Magnified feelings
☐ Irritability
☐ Cravings and urges
☐ Not interested in treatment
☐ Not participating in meetings
☐ Canceling appointments
☐ Relationship problems
☐ Other negative thoughts/feelings/actions

Client Worksheet 9

What About Alcohol?

You came to treatment because you have a problem with stimulants. You made a commitment to stop using stimulants. But you may not have made a commitment to stop using alcohol, especially if you have never had any problems with it. At the same time, people in your recovery program and in your 12-Step group may be putting a lot of pressure on you for complete abstinence from all drugs, including alcohol. Why should you stop using alcohol?

Using alcohol masks your emotions and feelings and does not allow you to fully experience them

- In what ways have you used alcohol to diminish certain feelings, avoid certain feelings, or change the way that you feel?

Using alcohol can arouse stimulant cravings

- During the last few times that you had a drink, in what ways did drinking arouse cravings for stimulants?

Using alcohol reduces your ability to resist stimulant cravings

- Describe some of the times that you thought you would enjoy having a drink but ended up experiencing cravings and urges for stimulants.

Using alcohol can lead to irresponsible and inappropriate behavior

- In what ways have you embarrassed yourself or experienced personality changes when using alcohol?

Using alcohol keeps you in contact with people, places, and situations that trigger stimulant cravings

- In what ways has using alcohol kept you in contact with people, places, and situations associated with stimulant use?

Adapted with permission from Washton, 1990b.

Client Worksheet 10

Action Plan for the Holidays

Are you the kind of person who normally looks forward to and enjoys the holidays? Or do you typically hope that the holidays pass by quickly? Either way, the holidays and other special events can be high-risk situations.

Times of celebration

For many people, the holidays are a time of fun and family. Although they may be fun, the holidays often involve parties with alcohol, intense involvement with family members, and time off from work. The desire to spend time with family and friends can seem like a good excuse to skip treatment and recovery meetings. Time off from work can turn into periods of boredom and restlessness, or isolation. The parties can be fun but filled with reminders of substance use as well as actual use. Overall, it can be an intense time.

Times of sadness

For many people, the holidays are reminders of the problems in their lives. Christmas, Chanukah, and Kwanza, which are family-oriented holidays, may be emotionally difficult times for people who are single, divorced, or in broken families. The holidays can provoke intense memories from childhood. These holidays and New Year's Eve can prompt some people to focus on what they consider their failures over the past year. Overall, it can be an emotionally stressful time.

- Alcohol and other drugs can be plentiful at holiday parties. What are the specific situations that you can expect to encounter this year? What happened last year? What specific steps can you take to address this problem?

- In what ways can the recovery-related routines of your life become disrupted during the holidays? What steps can you take to deal with this?

- During the holidays, do you have a lot of family members around? If so, in what ways can your family members interfere with your recovery? What can you do to strengthen your recovery routines and perhaps involve your family members in them?

- If you don't have a lot of family members around during the holidays, how can that be a problem? What steps can you take to strengthen your recovery routine during the holidays?

- Do the holidays represent to you times of intense activity or boring isolation? What can you do to help make the holidays as normal as possible? What kind of recovery-related activities can you plan to do?

Client Worksheet 11

Evaluating Your Self-Efficacy Regarding Relapse

An important lesson to be learned during recovery is to avoid high-risk situations whenever possible. Thus, one of your most important goals during recovery is learning how to avoid situations that are high risks for triggers, cravings, and relapse. However, not all high-risk situations can be avoided. You may run into your old dealer or drug-using friends, or someone at work may offer you drugs.

Because you will not be able to avoid all high-risk situations, another important goal during recovery is learning to respond to high-risk situations and preparing yourself for these. A part of this goal is evaluating your ability to handle these emergencies. The feeling that you can handle certain high-risk situations and prevent relapse is called "self-efficacy."

On one hand, it is foolish to believe that you can handle all high-risk situations or that you can handle any high-risk situations without first developing skills and tools to avoid relapse. On the other hand, it is equally foolish to believe that you cannot develop skills and techniques to handle high-risk situations. The task is to evaluate how you think you can handle certain situations that you are likely to encounter.

- Describe a high-risk situation that you encountered since starting treatment. How did you handle the situation? What happened?

- How would you rate your ability to handle the situation? Were you unsure and fearful? Were you certain and confident? Were you somewhere in the middle?

- What do you wish you had done? What would you do if the same situation happened today? Do you feel that you have made some progress in learning to deal with high-risk situations?

- What do you feel that you need to learn about to increase your ability to handle high-risk situations?

Client Worksheet 12

Increasing Your Self-Efficacy

Self-efficacy regarding relapse is the belief that you have developed the skills to handle certain high-risk situations. This usually involves having specific action plans to (1) refuse going to an even higher risk situation, (2) refuse offers of alcohol or other drugs, (3) leave the high-risk situation, (4) defuse the trigger by engaging in some activity, (5) speak with a sponsor or recovering friend, and (6) process the situation in a 12-Step or recovery meeting.

You can increase your self-efficacy in dealing with high-risk situations through experiences in real life as well as through role-playing exercises. You may discover that you are over-confident and need to develop more tools. Or you may discover that you have more tools than you thought.

Role-playing exercises

In the following role-playing exercises, the counselor will play "the other person." In each of these exercises, think about the action plan steps above mentioned above and imagine yourself really being in the situation.

- The phone rings. It is 6:00 p.m. It is the person from whom you have typically obtained your stimulants. He said that he called to see if you needed anything. Up to this point, you have not told him that you were in recovery.

- The phone rings. It is a friend with whom you have used stimulants for several months. You told him that you had stopped using stimulants but you knew that he still used. He asks if you want to go to the neighborhood bar and watch Monday Night Football.

- Your next-door neighbor is having a small party to celebrate graduating from college. You accept an invitation to attend. While you are there, someone whom you don't know well but who knows you from the neighborhood asks you if you want to go "for a walk" and smoke some marijuana.

Self-evaluation

For each of these role-playing exercises, describe how you felt regarding

- **Effectiveness:** Do you think that what you said in the role-playing exercise would be effective in the real world? What do you think was effective? What do you think needs work?

- **Confidence:** Do you feel confident to deal with this type of situation in real life? About what aspects do you feel confident? About what aspects do you feel that you need more work?

- **Action plan:** What specific action plan steps did you mention in the exercise? What action plan steps did you forget?

Client Worksheet 13

Stress: Identifying Your Warning Signs

Stress, anxiety, and anger are strongly connected to the ways in which you think and feel. They are also strongly connected to your physical well-being. That is, your experience of stress is related to the ways in which you think and perceive; they cause strong emotional responses, and they affect your physical well-being.

Above all, stress, anxiety, and anger are warning signs. They are ways that your body alerts you to the fact that something is wrong. They may not tell you exactly what is wrong, but they are warning signs that something needs to be changed.

When you are involved in stimulant use, it becomes easy to ignore these warning signs. An important task of recovery is to learn ways to decrease the levels of stress, anxiety, and anger in your life. But in order to do so, you must first learn to identify your warning signs. To help you accomplish this task, check off the following that have applied to you since being in treatment. Discuss what was going on in your life shortly before and while experiencing these warning signs of stress.

☐ Feeling anxious, nervous, fearful, or afraid. When do you have these feelings?

☐ Worrying about what might happen; imagining the worst. About what?

☐ Feeling irritable, cranky, and moody. When? What days and times?

☐ Feeling overly stimulated and distressed by the things around you. When? Where?

☐ Feeling angry, annoyed, and combative. When? Where? With whom?

☐ Feeling restless, impatient, and fidgety. When? What days and times?

☐ Experiencing tension in your muscles. Where? When?

☐ Experiencing stomach aches, cramps, diarrhea. When?

☐ Feeling exhausted, weary, and fatigued. When?

☐ Having problems concentrating and following what you read or hear.

☐ Having problems falling or staying asleep or having restless sleep.

Client Worksheet 14

Anger: Identifying Your Warning Signs

Physical signs of anger

Because the physical signs of anger are caused by a part of your nervous system, they happen automatically. During an episode, you may have a few or all of these signs. They are temporary and will rapidly fade if you allow yourself an opportunity to cool down.

- When you are angry, the pupils of your eyes can open up to let in more light. Have you ever noticed that it suddenly seemed brighter than before when you were angry?
- When you get angry, can you feel your heart beating faster and harder than normal?
- Do you remember your breathing becoming faster and harder than normal?
- When angry, your blood sugar level can rapidly rise. Have you ever noticed that when angry, you suddenly have a lot of energy and feel like doing something physical?
- When you are really angry, your body produces extra sweat to cool you off. Have you ever noticed that you became sweaty and had clammy hands when you were angry?
- When you are angry, do you feel your muscles becoming tense, perhaps especially in your face or hands? Do you become red in the face? Do you become suddenly hot or cold? Do you get a knot in your stomach?

Emotional signs of anger

People have different emotional experiences when they are angry. Some people feel inadequate and insecure whereas others become aggressive and hostile. Others feel victimized.

- When was the last time that you were angry? During that episode, how did you feel? What were you thinking?

Behavioral signs of anger

People have different behavioral reactions to anger. Some explode in fits of rage and yell at or hit other people. Some people become silent and go off to be alone.

- During the last time that you were angry, what did you do? What did you say to others? How did that make you feel at the time? How do you feel about that now?

Situations associated with anger

It is important to examine the situations that seem to be associated with your getting angry. You may be able to identify certain patterns and learn to avoid them.

- What was going on during the past few times that you were angry? What happened?

Client Worksheet 15

Recovery-Related Stress Reducers

Life is full of *stressors*, or things in the world that put some type of pressure on us to do something, to act in a certain way, or to follow a rule. But *stress* is our internal response to stressors. For example, being caught in a traffic jam is a stressor. Getting angry, agitated, and anxious is a response to being caught in the traffic jam. That's stress. However, there are things that can stop the cycle of stressors becoming stressful experiences. These can be important aspects of your recovery.

Broadcast your recovery

If you are typical, a lot of people know that you use stimulants. Probably more than you realize. If you try to go through treatment without telling people, they will assume that you are still using. Your stimulant-using friends will call, drop by, and contact you to get together. That can cause a lot of stress. But if you broadcast your recovery, these people may stay away, and nonusing friends and family can help support your recovery. That can reduce your stress.

- Which people have you been reluctant to tell about your recovery? Why have you been reluctant? What benefit would there be in telling them? What steps can you take to do so?

Let go of certain things

Being in recovery means starting many new activities, such as attending group therapy and 12-Step meetings, and exercising. Because there are only so many hours in a day, some activities will have to stop. The goal is to let go of unhealthy and unproductive routines and replace them with healthy ones. Therefore, you must make priorities about which activities you must let go. Prioritizing your daily activities can reduce your stress.

- What are some healthy activities that you should add to your daily or weekly routine? Why have you been reluctant to add them? What would you have to stop doing? What specific steps must you take in order to start engaging in these activities?

Don't recover alone

Many people feel alone during certain phases of recovery. It is easy to focus on past mistakes and problems and feel depressed and anxious. But the focus of recovery is being with others, talking to others about your struggles and successes, and listening to others regarding the ways that they are getting healthy. The alternative is being alone, whether physically or emotionally. That's stressful. Make recovery-related and recreational-related plans with others. That can reduce stress.

- What are some examples in which you shut out others and don't let them become part of your recovery program? Why have you done this? What steps can you take to recover with others rather than alone?

Client Worksheet 16

Learning To Solve Problems

During periods of active stimulant use, people are often overwhelmed by drug-related problems. They often hope that problems will disappear. When ignored, problems tend to worsen and become more numerous. The good news is that problems can be solved. But the solution involves an action plan.

Identify one problem at a time

Stimulant-related problems can seem overwhelming and prompt strong emotions. Focus on one problem at a time.

- Which of your stimulant-related problems seems to be the most important for you to address?

Understand the problem

- How did the problem get started? What made it get worse? How can you avoid it in the future?

- What is the nature of the problem? What did you do to cause it or make it worse?

Identify potential solutions

Write down all the potential solutions you can think of, especially those that you haven't tried before. Get advice from others.

- What are some solutions that worked in the past? What might happen if you use these solutions?

Make an action plan

Identify one solution that you feel might work. Make a plan to put the solution into action. What should you do? When should you do it? Who can help you?

- What are the specific steps that you need to take to get the job done?

Don't give up

If the problem is not solved as you had hoped, do not give up. See if you can figure out why the solution did not work. Was there something that worked partially but not completely? Identify new potential solutions and make another action plan. Most important, don't procrastinate and ignore the problem.

Client Worksheet 17

Managing Your Anger

People get angry. It's a part of life. You will become angry at various times in your life. However, it is unhealthy to remain angry. Anger can make you impulsive, prompting you to say and do things that you don't mean to do. Anger can be a trigger for stimulant cravings.

You will become angry during your recovery. You will think about situations associated with deep-seated emotions. You will be expected to talk about feelings that you have never discussed with anyone before. As you become increasingly aware of the negative consequences of your stimulant use, you may experience strong emotions that are uncomfortable.

You probably learned unhealthy ways to deal with anger. You may tend to repress your anger and pretend that everything is okay. You may impulsively explode and express your anger through physical or emotional abuse of others. Or you may let it build up and let it eat away at you. However, you can learn to manage and express your anger in healthy ways.

- **Identify your anger signs.** What are the physical, emotional, and behavioral warning signs of anger? Do your muscles get tense? Do you clench your fist or teeth? Do you become irritable, nervous, or short tempered?

- **Evaluate the situation.** Why are you angry? Are you overreacting to a real situation? Are you reacting to a situation that may never happen? Are you angry about one thing but expressing anger about another? Are you feeling impulsive, threatened, or scared?

- **Talk it out.** Rapidly identify someone with whom you can talk about the problem. Who do you know whom you can call immediately? Can you call your sponsor or a friend in recovery? Make plans now to attend the next 12-Step or recovery group meeting to talk about it. Identify someone who is not part of the problem.

- **Cool off.** It is better to talk out your anger rather than to act on it. Whether or not you can rapidly find someone with whom you can talk, take a break from the situation and cool off. Before you act on your anger, take a walk, do some exercise, do something physical, or take a shower.

- **Wait before responding.** If the problem is such that you have to respond, then don't respond until your physical and emotional signs of anger have gone away. Don't make a decision while feeling the signs of anger. Can you wait an hour? Can you wait a day? It will give you time to organize your thoughts and review your options. Talk with the person with whom you are angry only after you cool off.

- **Review options for action.** If you must respond, review your options. What has worked in the past? What option would be best for your recovery?

Client Worksheet 18

Selective Memory About Stimulant Use

There is no question that stimulants can provide a dramatically euphoric experience that is outside of normal human experiences. That is one of the reasons why people use stimulants. You probably had several stimulant-induced experiences during which you felt intensely euphoric, extremely powerful, and invulnerable. However, these experiences are accompanied by equally strong but negative experiences.

You also had many experiences during which you felt extremely depressed, agitated, and irritable. Similarly, you also experienced many adverse consequences related to your stimulant use, such as financial and employment problems, problems with family and friends, health-related concerns, and legal problems.

It is natural to focus on the positive side of things and to want to downplay the negative side. In certain areas of life, that is a good rule to live by. But one of the tasks of recovery is to always remember the negative consequences caused by your stimulant use. One of the signs of relapse is when people selectively remember only the good times associated with stimulant use, especially the euphoric experiences. People who tell "war stories" and focus on their wild stimulant-related experiences can make themselves and the people listening to them experience triggers, cravings, and urges for stimulants.

- Do you sometimes catch yourself either fantasizing or talking about "how good things were" back when you used stimulants? When you did this, what were you feeling? How did you feel afterward?

- Have you ever heard someone give a "drugalogue" or a "war story" about his wild experiences using stimulants and about all the positive but none of the negative aspects? How did it make you feel? Why do you think that people do this?

- Some people tend to have vivid memories of the euphoric effects of stimulants during certain times, such as when they feel overwhelmed, when they don't feel good, or when they are exhausted. Have you ever fantasized or talked about the euphoric effects of stimulants during times when you were not feeling good?

Client Worksheet 19

Fantasies About Controlled Use

After being in recovery for several weeks or months, you generally start feeling better. Although the healing process is just beginning, your thinking begins to be somewhat more clear, you are learning to experience and express your feelings more effectively, and you are learning problem-solving skills. A few or many of the negative consequences of your stimulant use are becoming less severe and numerous.

It is during these early recovery phases that you may have fantasies about being able to return to stimulant use. You may believe that if you made some changes, you could once again use stimulants. You may tell yourself that if you are "careful" you could use stimulants without losing control. You may believe that you are ready to try using stimulants "one last time" to test whether you can use stimulants without losing control over its use. These are called "fantasies of controlled use." They are classic warning signs of impending relapse.

If you experience fantasies of controlled use, you should immediately develop an action plan. This plan should include: (1) recognizing these as fantasies and rejecting them as options; (2) recognizing these as warning signs of impending danger; (3) immediately seeking a 12-Step sponsor, a counselor, or a recovering friend to speak with; (4) attending a 12-Step and recovery group meeting as quickly as possible; and (5) talking about these warning signs at the meetings.

- Have you ever thought about how nice it would be to use stimulants without all the adverse consequences?

- Have you ever thought about how good it would be to use stimulants but not lose control over them?

- Have you ever used stimulants without eventually losing control, such as losing control over the amount of the drugs, the amount of money or time spent on the drugs, or on your stimulant-induced behavior?

- When you used stimulants in the past, what were some of the most troubling problems that you experienced as a result of your stimulant use?

- What is your action plan for dealing with the warning sign of fantasies of controlled use? What are the specific steps of your action plan?

Client Worksheet 20

Those Ugly Reminders

If you find yourself thinking about those "great" times when you used stimulants or fantasizing about being able to control your stimulant use, you may benefit from what can be called "ugly reminders" about your stimulant use. These can help you to remember that your stimulant use included some fairly serious negative consequences and problems.

But don't go overboard. It is important to remember the kinds of problems that your stimulant use caused. You don't, however, want to beat up on yourself. Rather, when you experience the warning signs of selective memory and fantasies of control, remind yourself about the dark side of your stimulant use.

- Make a list of all the people who were hurt as the result of your stimulant use. List the friendships that were damaged as a result of your stimulant use. Put down friends' names and mention at least one specific way in which they were hurt because of your stimulant use. When you feel cravings, fantasize about controlled use, or begin to daydream about how nice things were when you used stimulants, review the names on this list and call one of them.

- Do you have some visible reminder of the negative consequences of your stimulant use? This can include an eviction notice, a job termination notice, divorce papers, a car repair bill, notices from bill collectors, or a hospital bill. Take one of these and put it inside a table-top picture frame. Whenever you feel cravings, have selective memories about the positive aspects of stimulant use, or fantasize about controlled use, put the ugly reminder where you will see it for the next couple of hours.

- Make a list of all of the things that you lost as a result of your stimulant use. These can include lost jobs, opportunities for advancement, relationships, cars, money, and memories. Keep this list handy and look at it when you begin having cravings or thinking about the stimulant highs.

Client Worksheet 21

Recreational and Leisure Activities

When you were using stimulants, there were times when your life focused on obtaining, using, and recovering from the effects of the drugs. There were times when your life was probably chaotic, out of control, and without structure. Recovery is an opportunity for you to develop a structure to your life and fill it with healthy activities.

Recreational activities are experiences in which you actively participate in an organized activity, generally with others, to have fun and enjoy life. They include participation in sports, arts and crafts endeavors, and table games, as well as sober dances, bowling, touch football, and card games. Some involve physical exercise. *Leisure activities* are things that you do primarily for relaxation and pleasure, and which don't involve much work. These include taking walks, having friendly conversations, reading books, watching movies, or watching sports activities.

- Before you began using stimulants, what type of recreational activities did you enjoy? What type of leisure activities did you enjoy? What were some of the benefits that you obtained from these activities?

- On Client Worksheet 22, *Examples of Recreational and Leisure Activities*, there are lists of recreational activities. Which of these do you find interesting? What are some of the benefits that you might obtain from these activities? In what ways could you incorporate these into your recovery program?

- On Client Worksheet 22 there are lists of leisure activities. Which of these do you find interesting? What are some of the benefits that you might obtain from these activities? In what ways could you incorporate these into your recovery program?

- Do you have any healthy hobbies? Do you see anything on Client Worksheet 22 that grabs your attention? Can you think of something else that would be personally satisfying?

Action plan

- Which recreational and leisure activities would you like to do within the next couple of weeks? Where would you go? When would you go? With whom would you go?

- What obstacles do you think you might encounter? What do you need to do to overcome these obstacles?

- What steps do you need to take in order to engage in these activities? Do you need help to organize the activities? When can you take the first step?

Client Worksheet 22

Examples of Recreational and Leisure Activities

Recreational activities, leisure activities, and hobbies are all ways in which you can have fun, enjoy being with others, and add healthy activities to your life. They can also help you take your mind off drugs, add structure to your life, and maybe even learn something new. They can help you avoid being bored and restless. They can help you reduce stress and anxiety.

Recreational Activities

Backpacking
Baseball/softball
Basketball
Billiards/playing pool
Bowling
Camping
Canoeing
Checkers
Chess
Dancing
Golf
Ice skating
Playing cards
Sailing/boating
Shuffleboard
Skiing
Skindiving
Surfboarding
Swimming
Table tennis
Touch football
Volleyball
Weightlifting
Other: ____________

Leisure Activities

Attending auctions
Attending auto races
Attending concerts
Attending plays
Attending sports events
Bicycling
Bird watching
Coin collecting
Crossword puzzles
Dining out
Driving
Fishing
Hiking
Horseback riding
Listening to music
Painting
Picnics
Playing video games
Reading books
Roller skating
Sightseeing
Sunbathing
Talking to friends
Visiting museums
Walks in parks
Watching movies and TV
Writing
Other: ____________

Hobbies

Amateur radio
Aquarium making
Arts and crafts
Astronomy
Auto repairing
Carpentry
Ceramics/pottery
Coaching Little League
Computers
Cooking/baking
Electronics
Flower arranging
Gardening
Genealogy
Home decorating
Hunting
Model building
Photography
Playing music
Sewing
Singing
Stained glass making
Volunteering
Woodworking
Other: ____________

Client Worksheet 23

Exercise and Recovery

Some people find having a regular schedule of intense exercise workouts, such as aerobics and step-aerobics especially enjoyable. But exercise doesn't always have to be intense to be healthy and can be somewhat more gentle, such as vigorous walks, bike rides, and skating. Also, some people find it difficult to engage in exercise just for the sake of exercise. They may find greater satisfaction in exercise activities that are social and involve groups of people. This can include dancing, tennis, swimming, or having entire groups going for walks, taking bike rides, going for runs, and skating.

Some of the benefits of exercise include increasing your physical well-being, improving your emotional well-being, improving your mental alertness, improving your sleep, providing you with more energy, and reducing your stress and anxiety. Exercise also provides structure to your life and can help to prevent weight gain.

- When was the last time that you engaged in regular exercise? What kind of exercise have you been involved with in the past?

- What type of exercise appeals to you the most? Intense, vigorous workouts by yourself? Competing with others? Social and gentle aerobic activities?

- What would you like to gain from exercise? What is the primary thing that would motivate you to exercise?

- Look at Client Worksheet 24, *Types of Exercise Activities*. What specific types of exercise would you consider doing? What would be a reasonable exercise schedule that you could incorporate into your recovery program?

- What obstacles might get in the way of your engaging in regular exercise? What steps can you take to overcome these obstacles? Are there clubs or organizations that you could join?

Client Worksheet 24

Types of Exercise Activities

You may not have noticed, but there are numerous opportunities for participating in exercise activities nearby. Unless you live in a very rural area (and maybe even if you do), you probably live near a county or city recreation department, a local YMCA gym, a Jewish Community Center, and private exercise clubs and gyms. Many of these, especially gyms that are sponsored by non-profit organizations, offer services that are low-cost or even free. You can get local information through the yellow pages under "gyms," "exercise," "recreation," and by contacting the local city or county recreation department.

Traditional activities

- Jogging
- Walking
- Bicycling
- Skating
- Swimming
- Weightlifting
- Nautilus-type workouts

Exercise classes

- Aerobics classes
- Jazz-aerobics
- Low-impact aerobics
- High-impact aerobics
- Step-aerobics classes
- Water-aerobics

Martial arts

- Judo
- Jujitsu
- Karate
- Kung-Fu
- Tai-Chi

Sports-type exercise

- Baseball
- Basketball
- Racquetball
- Roller hockey
- Softball
- Soccer
- Tennis
- Volleyball

Dance classes

- Ballet dancing
- Ballroom dancing
- Country and western
- Ethnic dancing
- Jazz dancing
- Latin dancing
- Modern dancing
- Swing dancing
- Tap dancing

Client Worksheet 25

Nutritional Self-Assessment

If you are typical, your use of stimulants and other drugs, especially alcohol, has had a bad impact on your diet. Stimulants suppress your appetite by making you feel as if your hunger is satisfied, even though you have not eaten anything. When your appetite is artificially suppressed by stimulants, you eat less and therefore do not consume sufficient calories and nutrition. At the same time, stimulants speed up the metabolism of your body, creating an even greater need for calories. Also, if you typically use alcohol in combination with stimulants, you may have gotten most of your calories from alcohol, often called "empty calories."

In addition to not eating frequently enough, you may have learned poor ways of eating. For instance, you may have learned to eat impulsively. Also, you may have developed the habit of eating foods with little nutritional value when you did eat.

Nutritional self-assessment

- If you tended to use stimulants in binges, for how many days did you binge? During a typical stimulant binge, how many days would you go without eating? What was the longest period of time that you went without eating?
- During a typical stimulant binge, how many times did you actually stop and eat a meal? Did you frequently "forget" to stop and eat?
- During or following a stimulant binge, when you did eat, what did you eat? What would you eat for breakfast? What would you eat for lunch? What would you eat for dinner?
- When you did eat, did you make plans to prepare and eat meals or did you eat impulsively?
- Now, during your recovery, do you make plans to prepare and eat meals or do you eat "when you can?"
- What did you eat for breakfast today? What did you have for lunch today or yesterday? What did you have for dinner yesterday?
- Do you notice that there are times when you crave fats and sweets?

Client Worksheet 26

The Food Guide Pyramid

You probably remember reading something about the Food Guide Pyramid, a guide to daily food choices, although you may not have thought about how it applies to you. Reviewing these five food groups and incorporating this information into your life can be important aspects of your recovery. Eating regularly, and eating meals that are balanced among these food groups, can help to decrease stimulant cravings, increase sleep, increase concentration, decrease withdrawal-related anxiety and depression, and provide sufficient energy required for recovery. The following provides a basic description of the five food groups and the recommended number of servings per day for each food group. Keep in mind that a "serving" is actually a fairly small amount.

Fruit group and vegetable group

These are good sources of fiber and vitamins. Having sufficient fiber prevents constipation. Having sufficient vitamins ensures the healthy functioning of the brain, nerves, muscles, skin, and bones. Some vitamins help energy to be released from food. A healthy diet should include three to five servings of vegetables and between two and four servings of fruits each day. A serving can consist of ½ cup of fruit or vegetables, a small salad, one medium potato, or a wedge of lettuce.

Bread, rice, cereal, and pasta

These are good sources of protein, vitamins, and minerals. Proteins are the primary building blocks of muscle, skin, blood, and bones. The brain chemicals that become depleted by chronic stimulant use are made from proteins. A healthy diet should include between 6 and 11 servings from this group each day. A serving can consist of 1 slice of bread, ½ cup of pasta or rice, or 1 ounce of cereal.

Milk and cheese

These include milk, ice cream, yogurt, cheeses, and cottage cheese. These foods are a source of calcium, protein, and vitamins. Calcium is required for healthy bones and teeth. A healthy diet should include at least two to three servings each day. A serving can consist of 1 cup of skim milk, 1½ cups of natural cheese, 1½ cups of lowfat ice cream, or 1¼ ounces of hard cheese.

Meat, poultry, fish, dry beans, eggs, and nuts

These foods are rich in protein, minerals, and vitamins. A healthy diet should be limited to two to three servings per day from this food group. A serving can consist of 2 to 3 ounces of chicken, fish, or lean beef; 1 egg; ½ cup of cooked dry beans; ½ cup of nuts; or 2 tablespoons of peanut butter, which is equivalent to 1 ounce of lean meat.

Fats, oils, and sweets

No serving sizes are suggested because these foods should be eaten sparingly.

- Write out all of the foods that you ate yesterday. How many of the essential food groups did you consume yesterday?
- Which of the food groups do you need to increase? Which do you need to cut down? What steps do you need to take to make that happen?

Client Worksheet 27

My Nutrition Improvement Action Plan

Stimulant-addicted people learn to act on impulse. It becomes commonplace to not eat regularly, to eat on the run, and to select foods based only on taste and not nutritional value. There is often no set schedule for meals, no meal planning, and an overreliance on high-calorie, high-fat fast foods, such as hamburgers and fries. However, with a little planning, eating can be transformed from an impulsive activity to an important and healthy component of recovery.

Make a schedule and a commitment

It is important to stop eating on the run. Making a schedule for meals can be a simple but very effective way to help add structure to your day. If you live with family members, mealtime can be a point during which all family members come together at least briefly. Take the time to list your daily and weekly priorities, such as 12-Step and recovery group meetings, and make a schedule that includes both meals and recovery priorities. Then make a commitment to continue and update this pattern.

Plan a few meals

It may seem foolish at first, but take the time to reflect on what meals you would like to have over the next several days or week. You don't have to plan out each meal. Rather, make a decision about some of the meals that you would like to have over the next several days, especially for dinner. In this way, you can plan ahead and purchase only those grocery items that you need to make the meals.

Make a grocery list

Once you have decided which meals you would like to have over the next several days, take the time to make a list of the grocery items that you need to prepare the meals. This helps you to avoid walking around the grocery store without a plan and buying groceries impulsively. Also, it will save money. You can divide your list into breakfast items, lunch items, dinner items, and snacks. Consider getting fruit as the primary type of snacks.

Plan meal preparation

Many people don't like to cook or clean up. If you live with others, it can be valuable to make a schedule about who does what. You can make agreements with others so that if one person cooks, another person cleans up.

Plan exceptions to the schedule

Most people enjoy eating out from time to time. You may have a favorite restaurant. However, eating out is often an impulsive behavior learned during periods of stimulant use. Thus, you can learn to incorporate eating out into your weekly schedule. In this way, eating out can be seen as a treat or a reward for keeping a healthy dining schedule.

Client Worksheet 28

Sample Behavioral Contract for Stimulant Abstinence

This is an agreement between ____________________ (the client) and ____________________ (the clinician) to help ____________________ (the client) maintain abstinence from stimulants.

I request my counselor to establish a schedule for collecting urine specimens from me for 24 weeks. I will provide urine samples three times per week on a Monday, Wednesday, and Friday schedule during the first 12 weeks of treatment. During the second 12 weeks (weeks 13 through 24), urine samples will be collected two times per week on a Monday-and-Thursday schedule. A clinical staff member of my sex will observe the urination. Half of each urine sample will be submitted for immediate analysis, and half will be saved at the clinic. Samples will be assayed for a variety of drugs of abuse, among which are cocaine, amphetamines, opioid drugs, marijuana, and sedatives. Each specimen for the collection request will consist of 3 ounces of urine. If the quantity is insufficient for analysis, that shall be considered a failure to provide a scheduled sample.

If I travel out of town due to an emergency, I will inform my therapist in advance of leaving. My therapist is authorized to verify such absences with ________________________ [significant other, etc.]. If I require hospitalization, my therapist will arrange to collect urine samples in the hospital. If I am sick and do not require hospitalization, I will still arrange to produce scheduled urine specimens. If I have difficulties with transportation, or inclement weather makes it difficult to travel, I will arrange (with the help of clinical staff) a way to get to the clinic for urine collection. On certain major holidays, the clinic will be closed. My therapist and I will mutually agree to modifications of the urine testing schedule during holiday weeks.

If for appropriate medical reasons, I am prescribed medication that is also a drug of abuse, I will provide to my therapist the name and phone number of my physician or dentist. I hereby give my therapist permission to contact my physician or dentist by phone and mail if I am given such a prescription. I agree to provide to my therapist a photocopy of the prescription or permit my therapist to see the prescription container. If the medication is appropriately prescribed, the appearance of the drug in urine tests will not be counted as relapse to drug use.

Stimulant-free urine samples

For each stimulant-negative urine sample collected during weeks 1 through 12 of treatment, ______________ points will be earned. A voucher stating the earned point value will be presented to me following the collection of a stimulant-free sample. This voucher will specify the number of points earned for that day, as well as the cumulative points earned to date and their monetary equivalent.

During the first 12 weeks of treatment, the first stimulant-free urine sample will be worth 10 points, with each consecutive stimulant-free sample collected thereafter earning an increment of 5 points above the previously earned amount. For example, if 10 points are earned on Wednesday for a stimulant-free sample, Friday's stimulant-free sample will earn 15 points, Monday's will earn 20 points, and so on. As an added incentive to remain abstinent from stimulants, a ____________ bonus will be earned for each week of three consecutive stimulant-negative urine samples collected. Assuming there are no stimulant-positive urine samples collected, the ____________ bonus can be earned during the first 12 weeks of treatment. During the second 12 weeks of treatment, the incentive program will be changed. Rather than earning points for stimulant-negative samples, ____________ will be earned.

For the entire 24 weeks of treatment, immediately after the urinalysis test results indicate that the urine sample is stimulant-negative, the following will happen. The ______________ [positive incentive] (weeks 1 through 12) or ____________ [positive incentive] (weeks 13 through 24) will be delivered.

Stimulant-positive urine samples

All urine samples will be screened for drug use. A record will be kept of all drugs that screened positive, although this contract will be in effect for stimulants only. For each stimulant-positive urine sample, I will not receive ____________ [positive contingency].

Failure to provide urine samples

The failure to provide a urine sample on the designated date without prior approval from my therapist will be treated as a stimulant-positive sample, and the procedure above will be in effect.

My signature below acknowledges that I have read, understand, and agree to the conditions of this urinalysis monitoring process. This process has been carefully explained to me, and I understand the consequences related to providing both stimulant-positive and stimulant-negative samples while I am a client at the program.

________________________ ________________________

(Client) (Date)

________________________ ________________________

(Counselor) (Date)

Client Worksheet 29

Components of a Functional Analysis

A functional analysis is a technique that can help you to understand your stimulant use so that you can engage in problem-solving solutions that will reduce the probability of future stimulant use. It allows you to identify the immediate causes of your stimulant use. A functional analysis is a method that helps you examine three aspects of your stimulant use:

- The types of circumstances, situations, thoughts, and feelings that increase the likelihood that you will use stimulants (triggers)
- The positive, immediate, and short-term consequences of your stimulant use
- The negative and often delayed consequences of your stimulant use.

Triggers

In general, triggers are those circumstances, situations, people, locations, thoughts, and feelings that increase the likelihood that you will use stimulants. They do not force you to use stimulants, but they increase the likelihood that you will use them.

Feelings and thoughts

When you encounter a trigger, you typically respond with certain thoughts and feelings regarding the immediate consequences of using stimulants, such as feeling better, having fun, or forgetting about troubles. You may think about the steps that you need to take to obtain and use stimulants.

Behaviors

Once you are exposed to triggers, and after you start having thoughts and feelings about stimulants, you engage in certain behaviors. One of those behaviors is using stimulants. However, through treatment, your stimulant use can be replaced with alternate coping behaviors.

Positive consequences

Almost immediately after using stimulants, you experience positive, strongly reinforcing consequences. Some of the positive consequences include feeling euphoric, having more energy, feeling more sexual, forgetting negative events or feelings, not feeling sadness or depression, or not feeling emotional pain. These positive consequences are generally immediate and short-term.

Negative consequences

Some of the negative consequences are experienced during or shortly after stimulant use episodes, such as spending too much money, engaging in high-risk sexual behavior, irritating or injuring others, or missing work or school. Many of the negative consequences are delayed or take a while to develop, such as damage to family and social relations, financial health, emotional health, physical health, educational goals, vocational stability, and legal status.

Client Worksheet 30

Preparing To Conduct a Functional Analysis: Identifying Your Triggers

This worksheet should be completed before using Client Worksheet 32, *Conducting a Functional Analysis of Your Stimulant Use,* and Client Worksheet 31, *The Functional Analysis Worksheet.* This worksheet will help you to identify the circumstances, situations, people, locations, thoughts, and feelings that increase the likelihood that you will use stimulants.

- List the places where you frequently used stimulants:

- List the people with whom you frequently used or purchased stimulants:

- List the times or days when you most frequently used stimulants:

- List the kinds of activities in which you were typically engaged when you used stimulants:

- List the feelings and emotions that you experienced after you were exposed to triggers:

- List the kind of things that you were thinking about after you were exposed to triggers:

Client Worksheet 31

The Functional Analysis Worksheet

Trigger	Feelings and Thoughts	Your Behavior	Positive Consequences	Negative Consequences

Client Worksheet 32

Conducting a Functional Analysis of Your Stimulant Use

This worksheet is used in combination with Client Worksheet 31, *The Functional Analysis Worksheet.* It should be used only after completing Client Worksheet 30, *Preparing to Conduct a Functional Analysis: Identifying Your Triggers.*

Step 1

On the *Functional Analysis Worksheet,* in the column titled "Your behavior," briefly describe an example in which you recently used stimulants.

Step 2

Think about what you were doing immediately prior to this episode of stimulant use. Can you remember who you were with, what you were doing, or the time of day? Place these in the "Trigger" column.

Step 3

Immediately prior to using stimulants during this episode, what were you thinking about? Do you remember what you were feeling? Place whatever thoughts and feelings that you can remember in the "Feelings and thoughts" column.

Step 4

What happened immediately after you used the stimulants? How did your mood change? Did you feel euphoric or powerful? Did you feel that you had more energy or power than normal? Did you feel happy or not as depressed as before? Did you stop feeling bad about something?

Step 5

What have been the long-term consequences of this and other episodes of stimulant use? How has it affected your relationships with friends? How has it affected your family? How has it affected your work or school situation? How has it affected your financial situation? How has it affected your emotional health? How has it affected your physical health?

Return to Step 1

Describe another example of a relatively recent episode of stimulant use. Repeat all the steps as before. Repeat this until Client Worksheet 31, *The Functional Analysis Worksheet,* has been completely filled.

Client Worksheet 33

Functional Analysis: Important Points To Consider

You can quit

You can learn to stop using stimulants. Other people with stimulant problems have been able to learn how to stop using stimulants.

Stimulant abuse is a learned habit

It is important to begin thinking of your stimulant use as something you have learned to do. It is a learned habit. Learning how to stop using stimulants does not require that you understand exactly how your stimulant problem began. Blaming other persons, events, or circumstances does not help you learn how to stop. But what is effective is learning that your stimulant abuse is a problem that you can do something about.

The goal is to learn to stop using and start living

One goal of this treatment program is to help you learn how to stop using stimulants and other drugs. Another equally important goal is to help you learn how to live a drug-free lifestyle. You will obtain the most benefit from treatment if we can help you stop your stimulant use so that we can focus on helping you make other lifestyle changes that will promote long-term abstinence from stimulants.

Slips are not treatment failures

Mistakes are preventable and should be prevented. But mistakes happen. If you use stimulants during treatment, you should not view it as failure. Rather, such incidents can be used to help you learn more about your stimulant use so that you can more effectively learn to stop using completely. However, it does not give you permission to use stimulants.

Practice is important

You must learn to work on these new skills between treatment sessions. Learning and practicing new skills and behaviors is necessary. Talking about making changes is not sufficient to deal with high-risk situations. Rather, you learn by practice.

Client Worksheet 34

Self-Management Planning

By now, you have identified several of your triggers. You can organize them into categories, such as high-risk places, people, times of the day, activities, and feelings. This helps you to see that certain triggers are *external* and exist primarily in your environment (such as places) and some are *internal*, such as feelings and thoughts. Different triggers require different responses.

Avoiding triggers

Some triggers, such as certain high-risk places and people, can be avoided. This can involve taking alternate ways home so that you don't pass by your stimulant dealer's house, or not passing by bars and clubs that you frequently went to in the past.

Rearranging the environment

Although you don't have complete control over your entire environment, you have a lot of control over much of it, such as in your home. You can rid your home of stimulants, drug-using paraphernalia, and dealers' phone numbers. You can stop carrying cash, especially when you know that you will be passing by high-risk places.

Developing a new coping plan

You cannot avoid certain triggers. If cash or a certain family member is a trigger for you, it will not always be feasible to avoid these triggers. Thus, you must learn to prepare to encounter such triggers by developing new strategies or plans to help you to not use stimulants in such situations (such as calling your spouse after handling cash).

Make several copies of Client Worksheet 35, *Self-Management Planning Worksheet*. For each worksheet, you will address one trigger. Engage in the following steps.

Step 1

Select a specific trigger that you need to address. It should be a trigger that you are likely to encounter before the next session. Write this down in the "Trigger" column.

Step 2

Think about the different ways in which you can deal with this trigger. Can you avoid the trigger? Can you rearrange your environment so that you don't have to encounter the trigger? Is there some new coping strategy that you can engage in the event that you do encounter the trigger? Write these down in the "Plans" column. You may have several plans for each trigger.

Step 3

Working with your counselor, consider the overall effects or consequences of each plan. Write these down in the "Positive and Negative Consequences" column.

Step 4

How hard will it be to carry out each plan? With "1" being the least difficult, and "10" being the most difficult, write down the level of difficulty in the "Difficulty" column.

Step 5

Select a plan that seems to be reasonable. Working with your counselor, engage in role-playing exercises and practice engaging in this action plan.

Working with your counselor, you should repeat the above steps for at least three triggers in this session, and identify three additional triggers to work on before the next session.

Client Worksheet 35

Self-Management Planning Worksheet

Trigger	Plans	Positive and Negative Consequences	Difficulty (1–10)
	Plan 1:		
	Plan 2:		
	Plan 3:		
	Plan 4:		
	Plan 5:		
	Plan 6:		

Client Worksheet 36

Relationship Happiness Scale

This scale is intended to estimate your current happiness with your relationship in each of the ten areas listed below. Ask yourself the following question as you rate each area: How happy am I with my partner today in this area? Then circle the number that applies. Remember, you are indicating your current happiness. That is, it represents how you feel today. Also, do not let your feelings in one area influence the rating in another area.

	Completely Unhappy									**Completely Happy**
Household responsibilities	1	2	3	4	5	6	7	8	9	10
Rearing of children	1	2	3	4	5	6	7	8	9	10
Social activities	1	2	3	4	5	6	7	8	9	10
Money	1	2	3	4	5	6	7	8	9	10
Communication	1	2	3	4	5	6	7	8	9	10
Sex & affection	1	2	3	4	5	6	7	8	9	10
Academic or occupational progress	1	2	3	4	5	6	7	8	9	10
Personal independence	1	2	3	4	5	6	7	8	9	10
Partner's independence	1	2	3	4	5	6	7	8	9	10
General happiness	1	2	3	4	5	6	7	8	9	10

Name ______________________________

Date ______________

Adapted with permission from Azrin et al., 1973.

Client Worksheet 37

Daily Reminder To Be Nice

It is easy for partners to take each other for granted, especially when stimulant use is part of the relationship. This worksheet is a way to help remind you that there are some simple and effective things that you can do to help reverse certain negative behaviors that may have become habitual in your relationship. This worksheet can help to remind you to do a few nice things for your partner and to record how often you actually engage in these behaviors.

Topic	Date	Date	Date	Date	Date
Did you express appreciation at least once to your partner today?					
Did you compliment your partner at least once today?					
Did you give your partner any pleasant surprises today?					
Did you express visible affection to your partner at least once today?					
Did you spend some time devoting your complete attention to pleasant conversation with your partner?					
Did you initiate at least one of the pleasant conversations?					
Did you make any offer to help before being asked?					

Adapted with permission from Sisson and Azrin, 1989.

Client Worksheet 38

The Perfect Relationship

In each area listed below, write down the activities that would exist in what would be an ideal relationship for you. Be brief, specific, and positive about what you would like to occur.

Regarding "household responsibilities," I would like my partner to:

1. ____________________
2. ____________________
3. ____________________
4. ____________________

Regarding "child-rearing," I would like my partner to:

1. ____________________
2. ____________________
3. ____________________
4. ____________________

Regarding "social activities," I would like my partner to:

1. ____________________
2. ____________________
3. ____________________
4. ____________________

Regarding "independence," I would like my partner to:

1. ____________________
2. ____________________
3. ____________________
4. ____________________

Regarding "personal habits," I would like my partner to:

1. ____________________
2. ____________________
3. ____________________
4. ____________________

Regarding "managing money," I would like my partner to:

1. ______________________________
2. ______________________________
3. ______________________________
4. ______________________________

Adapted with permission from Sisson and Azrin, 1989.

Client Worksheet 39

Positive Requests

If you or your partner wants the other to make changes, the most effective way to accomplish this is by using positive communication. This is more effective and more pleasant than by negative communication, such as making demands, nagging the other person, or trying to order the other person to do something.

Engaging in positive communication is a skill, and it can be learned. It also takes practice. In the beginning, it may seem unnatural, but as you practice and incorporate it into your daily lives, it becomes natural.

The ways in which requests are made can be pleasant and will increase the likelihood that the requests will be fulfilled.

Be selfish

Don't be greedy, but don't be shy. Think about what would make you really happy. If it seems reasonable, ask for it.

Take the other person's point of view

Try to take the other person's point of view and understand how he or she feels. The other person may not recognize what you feel you need. The other person may not even realize that you are unhappy.

Take partial responsibility

When appropriate, accept partial responsibility regarding the current situation. You may never have expressed how important a specific situation is to you. Similarly, you may be equally responsible for the way a specific situation has evolved. For example, you may want your partner to become more involved in the children's homework. You may want to remind your partner that you have never expressed how important it is to you that both of you should help the kids with their homework. Also, you may want to state to your partner that you recognize that you haven't been spending sufficient time helping the kids with their homework, either.

Offer to help

Offer assistance to make it easier for your partner to fulfill your request.

Always try to say yes, if possible

Because you are going to be asking your partner to do things that will make you happy, you should be willing to do the same for your partner.

Compromise when necessary

Things will not always be black or white. There are times when it is best to compromise. Be willing to compromise so that both partners have something to gain.

Client Worksheet 40

Reciprocal Contract for Behavior Change

This contract is designed to assist you in achieving and maintaining positive changes in your relationship. During treatment, you will be asked to develop several of these contracts which will document reciprocal changes requested by you and your partner. By making a public commitment and placing it in writing, you are actively taking steps toward achieving and maintaining positive changes in your relationship.

I, ________________________, agree to make every effort possible to make the following changes at my partner's request. I understand that this change is very important to him/her and therefore is also very important to me.

Behavior change

____

____

I, ________________________, agree to make every effort possible to make the following changes at my partner's request. I understand that this change is very important to him/her and therefore is also very important to me.

Behavior change

____

____

This contract will continue throughout treatment unless a new contract is substituted or until one or both of the parties decides to terminate his or her participation.

Client ______________________ Date ____________

Partner ______________________ Date ____________

Counselor ______________________ Date ____________

Client Worksheet 41

Improving Communications

Be polite to your partner

When talking to your partner, use the same courteous words and tone you would use with a stranger or a coworker.

Express positive feelings

Let your partner know what you like about the things that he or she has done. Focus on successes as much as on things that are not going well.

Do something nice

Without being asked or without a special reason, do something that your partner would like or find special. Also, do it without expecting something in return.

Determine the importance of an issue before complaining

Ask yourself whether or not something is worth complaining about. Express complaints only about things that matter.

Choose an appropriate time

Choose settings and times that are conducive to a positive discussion. Don't do it when either of you is angry or doesn't have time.

Have a goal in mind

What are you trying to achieve? What are you looking for? Why do you want these changes? Are they reasonable or achievable?

Be specific about your complaints

Focus on one thing at a time. Have a specific example of the problem. Be prepared to tell your partner precisely what you would like him or her to do differently. Stay focused, and don't bring up other problems.

Request changes in a positive manner

In a positive way, tell your partner what is bothering you and what you would like to see changed. Avoid criticisms, put-downs, and assumptions about motives.

Prepare to compromise

Be prepared to discuss solutions that work for both of you. Don't declare ultimatums or dismiss your partner's ideas.

Client Worksheet 42

Disagreements and Fights

Expect to have disagreements

Disagreements are normal aspects of relationships, even healthy ones. People in relationships will not always agree on everything.

Some disagreements are not disagreements

Very often, what people characterize as disagreements are in fact examples of miscommunication or poor communication.

Miscommunications involve unexpected responses

Miscommunications happen when the message that you are trying to send to your partner provokes a response that you did not expect or intend for him or her to have.

Miscommunications involve poor expression

Miscommunications often result from not expressing yourself clearly, specifically, or completely. Don't assume that you know what your partner does or does not know. Provide reasons why you are complaining or making a request.

The problem may be the message

You may have conveyed a message that you did not intend by not saying what you really meant, leaving out information, or by providing nonverbal messages inconsistent with the verbal message.

Arguing and fighting

People can argue and fight because communication skills used in this approach are not being followed. For example, when people don't remain focused on a topic, when they try to bring up issues when angry or at inappropriate times, or when they are overly critical, a discussion can easily get out of control and become a fight or an argument.

Recognize your pattern of fighting

The first step of gaining control of fighting and arguing behavior is to recognize your pattern of fighting. Fights can be thought of as bringing up issues without discussion or resolution. You can make lists of the types of situations that typically result in fights with your partner.

Avoidance

Some couples rarely argue but avoid conflict by never talking about important issues. In such situations, one partner typically gives in all of the time or both become adept at ignoring issues when they arise. This avoidant style of communication usually results in one or both partners feeling resentful, unloved, not cared for, or unimportant. It is important to develop communication skills that help you to recognize the issues that are important to both of you and to communicate requests and complaints at appropriate times.

Recognize avoidance problems

Some of the clues that avoidance may be a problem in your relationship are: (1) believing that there is no conflict in your relationship, (2) having dull and routine conversations that leave you feeling not connected, (3) avoiding certain topics because they will start fights, and (4) feeling resentful toward your partner so that you do not want to do special favors.

Client Worksheet 43

Good and Poor Listening Skills

Good listening promotes effective communication

It is important to engage in active listening to your partner. Active listening involves trying to completely understand what your partner is trying to communicate, specifically understanding what your partner wants and what your partner is feeling. When you think that you understand what your partner is trying to communicate, you can summarize what you think he or she is communicating and ask if you understand it correctly. You can ask your partner to explain it in more detail, or to provide examples, or ask him or her to explain it differently. You can ask what your partner is specifically feeling right now.

Validate your partner's feelings

It is important for you to let your partner feel that you can understand how and why he or she might feel the way that he or she is feeling. That is, you can communicate to your partner that his or her feelings make sense. You may not necessarily agree with your partner, but you can convey to your partner that you understand his or her point of view. This is an important way for you to communicate the message that you care about your partner and you care about the way that your partner feels. If you are angry and cannot validate your partner's feelings at the moment, you can request a short break, cool off for a few moments, and return when you can do so.

Poor listening sends poor messages

When you listen poorly, you can convey messages to your partner that will interfere with good communication. Poor listening conveys to your partner such messages as (1) I am not interested in your opinions or feelings; (2) Your feelings are silly; (3) You are foolish to have these feelings; (4) Your feelings don't deserve my attention; (5) My opinions and feelings are more important than yours; (6) My opinions and feelings are more reasonable than yours.

Types of poor listening behaviors

Self-summarizing involves the continual restatement of a position over and over during a discussion. *Cross-complaining* occurs when the complaint of one partner is met by a complaint by the other rather than trying to solve the original problem. *Mind-reading* occurs when issues are avoided by one partner feeling and acting as if he or she knows how the other partner feels or what the other would like to do. This results in the second partner feeling unimportant, left out of decisions, and resentful. *Yes-butting* involves one partner responding to the other with a series of "Yes, but...." statements. This sends the message that you don't want to change or meet your partner's needs or to understand your partner's point of view. *Character assassination* involves making requests or comments that attack your partner's whole self, rather than specific problem behaviors or areas for change. *The complaining rut* describes a pattern of communication characterized by continual complaints without suggestions for change or alternatives and without noting positive behavior changes.

Client Worksheet 44

Recovery Self-Evaluation

For each of the following topics, rate how satisfied you are at this time.

	Very Dissatisfied								**Very Satisfied**
Job/School	1	2	3	4	5	6	7	8	9	10
Friendships	1	2	3	4	5	6	7	8	9	10
Family life	1	2	3	4	5	6	7	8	9	10
Leisure activities	1	2	3	4	5	6	7	8	9	10
Recreational activities	1	2	3	4	5	6	7	8	9	10
Stimulant use	1	2	3	4	5	6	7	8	9	10
Stimulant cravings	1	2	3	4	5	6	7	8	9	10
Alcohol/drug use	1	2	3	4	5	6	7	8	9	10
Alcohol/drug cravings	1	2	3	4	5	6	7	8	9	10
Self-esteem	1	2	3	4	5	6	7	8	9	10
Physical health	1	2	3	4	5	6	7	8	9	10
Emotional health	1	2	3	4	5	6	7	8	9	10
Sexual fulfillment	1	2	3	4	5	6	7	8	9	10
Spiritual well-being	1	2	3	4	5	6	7	8	9	10

- Which areas have you improved the most since you began treatment?
- What are the areas that you feel need the most improvement?
- What specific action plans do you plan to engage in to accomplish these goals?
- What areas of your treatment plan do you think need enhancement?

Adapted with permission from Rawson et al., 1995.

Appendix C
Screening Tests for Cognitive Impairments

The following list provides a number of tests that can be used to screen for cognitive impairments in stimulant users. The first two can generally be administered by counselors who are culturally competent for their client population. The other six tests should be administered and interpreted by a psychological testing specialist.

These tests can be administered quickly and easily and are used extensively in batteries created by neuro- and cognitive psychologists. Although the tests are very sensitive in revealing the existence of cognitive problems, particularly when several of the tests are administered in combination, they do not provide information on the exact nature or depth of the impairment. Positive screens should be referred to an appropriate collaborator (e.g., a neuropsychologist) for more extensive assessments.

Cognistat (Neurobehavioral Cognitive Status Examination)

This test is designed to assess intellectual functioning in five areas in adults 18 years of age and older. Administration of the test takes about 10 minutes for cognitively intact individuals and from 20 to 30 minutes for those who are cognitively impaired. Cognistat is available from:

Psychological Assessment Resources, Inc.
P.O. Box 998
Odessa, FL 33556
1-800-331-TEST

Brief Neuropsychological Cognitive Examination (BNCE)

This test is designed to evaluate the cognitive status of clients with psychiatric disorders or psychiatric manifestations of neurological disease. The reading level required for the test is minimal, and it is recommended for assessing mood disorders and substance use disorders. Both adolescents and adults can be tested in about 30 minutes. The BNCE is available from:

Western Psychological Services
12031 Wilshire Boulevard
Los Angeles, CA 90025-1251
1-800-648-8857

Backward Digital Span

This test is simply saying numbers at the rate of one per second and asking the client to repeat them backwards. For example, you might say "3, 8, 6" and would expect a response of "6, 8, 3." The examiner starts with three digits and goes up to nine digits. Three different number strings are presented at each level. If the client misses all three at a particular level, then the test is stopped. This test takes about 5 minutes to administer. It is a test of working memory capacity.

FAS Test of Verbal Fluency

The client is asked to say as many words (excluding proper names) that start with the letter *F* as she can in 1 minute. This is then repeated with the letter *A*, and again with the letter *S*.

Digit Symbol Subtest of the WAIS-R

This test taps the same thing as the Symbol Digit Modalities Test (i.e., the ability to manipulate simple information and pay attention). It is part of the revised Wechsler Adult Intelligence Scale (WAIS-R) and can be obtained from:

The Psychological Corporation
Harcourt Brace & Co.
15 East 26th Street
15th Floor
New York, NY 10010-1505
1-800-211-8378

Trail Making Test, Parts A and B

This test is very sensitive to brain function, is essentially connecting the dots, and takes about 5 minutes for both parts. The citation for the test is as follows:

Reitan, R.M. Validity of the trail making test as an indication of organic brain damage. *Perceptual and Motor Skills* 8:271–276, 1958.

Stroop Color Word Interference Test

Many versions of this test exist. It taps selective attention and the ability to ignore irrelevant information. A version can be obtained from either of the testing companies listed above.

Shipley-Hartford Tests of Vocabulary And Abstract Thinking

This is a short-form IQ test that takes a maximum of 20 minutes to administer. It is old but still widely used—particularly in the aging literature. The citation for the test is as follows:

> Shipley, W.C. A self-administering scale for measuring intellectual impairment and deterioration. *Journal of Psychology* 9:371–377, 1940.

Repeated Memory Test (RMT)

The RMT, which was developed by Sara L. Simon, Ph.D., has five different versions, each equivalent in word frequency and length. Because the test is so simple, giving it multiple times does not seem to involve a learning curve. Each test consists of 25 words each printed ¾ inches high on a 3-inch × 5-inch card.

The client is first told that she will be shown some words and that she will be asked to remember them. Then the words are presented to the subject, one at a time for 1 second each. When the client has seen all of the cards, there is an approximately 10-minute interval filled with distracter tasks. During this interval, other tests—such as the Digit Symbol, Trail Making A, and Trail Making B tests—may be administered.

The client is then given the recall test and told to write down any words that she remembers. The client is allowed as much time as she needs to complete the test. However, if 2 minutes have gone by without the client responding, suggest that she stop because no one remembers them all.

Next the client is given the recognition test and asked to circle the words that she remembers being shown.

A copy of the test follows, but for further information on the RMT, contact

Sara L. Simon, Ph.D.
Los Angeles Addiction Treatment Consortium
1001 W. Carson St., Suite U.
Torrance, CA 90502
310-224-4670
310-782-9140 (fax)

Scoring:

The recall test has two measures.
1. Number correct
2. Number of false positives (items remembered but not shown)

The recognition test has two measures.
1. Number correct
2. Number of false positives (items remembered but not shown)

A large number of false positives suggests that the patient was guessing and probably is having memory problems.

Test 1 Words

SAW

OWL

TIE

CAP

KITE

NAIL

PLUG

DEER

CHAIR

TRAIN

STOVE

CIGAR

GLOVE

SALTSHAKER

ANCHOR

MONKEY

CANNON

SWEATER

BICYCLE

OSTRICH

THIMBLE

AIRPLANE

CIGARETTE

WATERMELON

GARBAGE CAN

Recognition Test 1

Circle any items that were shown to you today during the testing session.

BASEBALL BAT
CLOWN
DESK
BROOM
CHAIR
BUS
TRAIN
WINDOW
SAW
ENVELOPE
KITE
THIMBLE
TREE
MOON
LEG
PLUG
CLOCK

FOOTBALL
MITTEN
BICYCLE
WATERMELON
OWL
DEER
PIPE
SWEATER
BELT
NAIL
CANNON
GARBAGE CAN
GRASSHOPPER
KANGAROO
BREAD
COAT
MUSHROOM

OSTRICH
SALTSHAKER
RUG
CHICKEN
ROOSTER
GLOVE
ANCHOR
GRAPES
STOVE
IRON
MONKEY
CIGAR
AIRPLANE
TIE
CIGARETTE
CAP
COW

Test 2 Words

RING

ANT

CAT

POT

CARROT

CORN

STAR

LION

LAMP

BEAR

PEACH

COUCH

CHAIN

CHURCH

BEETLE

BLOUSE

WRENCH

PEPPER

GORILLA

PEACOCK

UMBRELLA

SUITCASE

ACCORDION

TENNIS RACKET

CLOTHESPIN

Recognition Test 2

Circle any items that were shown to you today during the testing session.

ACCORDION
BEETLE
JACKET
HANGER
RACCOON
ROLLING PIN
TENNIS RACKET
GORILLA
UMBRELLA
MOTORCYCLE
CUP
SWING
FOOT
CORN
CAT
STAR
DRESSER

PEACH
PEPPER
EAGLE
CAMEL
WRENCH
POT
FENCE
ARM
SKIRT
CAR
ANT
SUITCASE
LION
CLOTHESPIN
LEAF
SOCK
CONCH

TOOTHBRUSH
CARROT
LETTUCE
BEAR
PLIERS
RABBIT
CHAIN
PEACOCK
SAILBOAT
LAMP
GIRAFFE
TELEPHONE
FOX
BLOUSE
CHURCH
RING
SHOE

Test 3 Words

TOP

PIG

EAR

LIPS

REFRIGERATOR

FISH

PAINTBRUSH

SHOE

SWAN

HORSE

RULER

CROWN

ONION

WATCH

PENCIL

BARREL

BASKET

DONKEY

TRUMPET

PENGUIN

CHISEL

SCISSORS

SNOWMAN

ALLIGATOR

ARTICHOKE

Recognition Test 3

Circle any items that were shown to you today during the testing session.

DOORKNOB
TOMATO
WATCH
CHISEL
POTATO
KETTLE
ASPARAGUS
FINGER
SLED
CROWN
SWAN
POCKETBOOK
BALL
KEY
DOLL
PIG
TOP

REFRIGERATOR
BARN
CELERY
PANTS
TRUMPET
SHEEP
PENCIL
HORSE
HAT
LOBSTER
WINEGLASS
NUT
RULER
FORK
PEANUT
EAR
FISH

ALLIGATOR
ARTICHOKE
HELICOPTER
PENGUIN
TABLE
SCISSORS
SNOWMAN
WINDMILL
SHOE
BARREL
DONKEY
BASKET
DOG
PAINTBRUSH
LIPS
ONION
BOAT

Test 4 Words

BED
TOE
COW
BOOK
GOAT
WHEEL
BELL
FLAG
DRESS
STOOL
SNAIL
MOUSE
THUMB

PIANO
BOTTLE
GUITAR
SPIDER
LEOPARD
PITCHER
BALLOON
SEA HORSE
RHINOCEROS
BUTTERFLY
LIGHT SWITCH
TOASTER

Recognition Test 4

Circle any items that were shown to you today during the testing session.

PITCHER
BUTTERFLY
LADDER
PUMPKIN
SCREWDRIVER
APPLE
BOTTLE
TOASTER
BANANA
SPOON
LEOPARD
SPIDER
HAIR
WHEEL
STOOL
SUN

LIGHT SWITCH
VIOLIN
ASHTRAY
SNAIL
SEA HORSE
BOOK
IRONING BOARD
DRESS
FLAG
LIGHTBULB
KNIFE
HEART
MOUSE
BOW
SHIRT
BELL

TOE
GUITAR
CAKE
BOWL
CANDLE
HAMMER
THUMB
ROLLERSKATE
AXE
BALLOON
PIANO
RHINOCEROS
BIRD
TIGER
BED
GOAT

Test 5 Words

EYE
BEE
FLY
GUN
HARP
DUCK
LOCK
LEMON
BRUSH
BUTTON
ARROW
SCREW
GLASS
WAGON
COMB
CHERRY
ORANGE
FLOWER
WHISTLE
FRYING PAN
SANDWICH
NECKLACE
PINEAPPLE
TELEVISION
CATERPILLAR

Recognition Test 5

Circle any items that were shown to you today during the testing session.

COMB
ORANGE
SANDWICH
FROG
TELEVISION
GLASS
CHERRY
FLOWER
ROCKING CHAIR
TRUCK
LEMON
BEE
SKUNK
SEAL
CLOUD
NECKLACE
DOOR

NAIL FILE
EYE
MOUNTAIN
WAGON
HARP
PEN
CATERPILLAR
NEEDLE
SNAKE
BUTTON
FRYING PAN
VEST
GUN
ELEPHANT
FLY
ARROW
VASE

PINEAPPLE
TURTLE
WHISTLE
SCREW
BOOT
TRAFFIC LIGHT
GLASSES
NOSE
SQUIRREL
STRAWBERRY
PEAR
FLUTE
BRUSH
DUCK
BOX
LOCK
SOFA

Name____ **Date** ______________

Recall Test for Words

Please write down all of the test words that you remember below:

Appendix D—Glossary

Addiction: A chronic, relapsing disease, characterized by compulsive drug-seeking and drug use, and by neurological adaptations in the brain.

Amygdala: A discrete brain area that is part of the limbic system, has a large number of dopamine-containing neurons, and plays a role in the learning and performing of certain behaviors in response to incentive stimuli (i.e., motivation, reinforcement).

Analog: A chemical compound that is similar to another drug in its effects, but differs slightly in its chemical structure.

Anergia: Lack of energy.

Anhedonia: Loss of interest in pleasurable activities; the inability to feel pleasure.

Anorexia: Loss of appetite, accompanied by weight loss and thin, gaunt appearance.

Arrhythmia: Irregular heartbeat.

Axon: A long, thin fiber that conducts electrical impulses away from the neuron's cell body and on to other neurons.

Benzodiazepines: Drugs that relieve anxiety or are prescribed as sedatives; they are among the most widely prescribed medications and include valium and librium.

Bradycardia: Slowed heartbeat.

Bruxism: The habitual, involuntary grinding of teeth, usually during sleep.

Cachexia: Weight loss, wasting of muscle, and debility.

Central nervous system (CNS): The brain and spinal cord.

Cerebellum: A brain structure that controls coordination and regulation of complex voluntary muscular movements, posture, and balance.

Choreoathetoid: Involuntary movement.

Convulsion: An abnormal, uncontrollably violent involuntary contraction or series of contractions of the muscles; spasm or series of jerkings of the face, trunk, or limbs.

Craving: A powerful, often uncontrollable desire for drugs.

Dendrites: Thin, branched extensions of a neuron that extend from the cell in branched tendrils to receive information from adjacent neurons; they conduct electrical impulses inward toward the cell body.

Dermatitis: Inflammation of the skin.

Designer drug: A synthetic analog of a restricted drug that has psychoactive properties.

Detoxification: A process of allowing the body to rid itself of a drug while managing the symptoms of withdrawal; often the first step in a drug treatment program.

Diaphoresis: Profuse sweating, often with chills.

Diastolic blood pressure: The pressure exerted by the blood on the cavities of the heart at the moment when they fill with blood.

Dopamine: A neurotransmitter present in several brain regions involved in movement, emotion, motivation, reinforcement, and feelings of pleasure.

Dopaminergic: Dopamine-mediated.

Dysphoria: A mood of general dissatisfaction, restlessness, and anxiety.

Glucose utilization: A general indicator of physiological activity; in the brain, an indicator of neurological activity presumed to be information processing.

Hypertension: Elevated blood pressure.

Hyperthermia: Elevated body temperature.

Limbic system: A group of subcortical brain structures that are especially concerned with emotion and motivation.

Narcolepsy: A disorder characterized by uncontrollable attacks of deep sleep.

Neuron: The morphological and functional unit of the nervous system, consisting of the cell body, dendrites, and axon.

Neurotransmitters: Chemical substances that transmit signals between neurons and that modulate neuronal activity.

Nucleus accumbens: A discrete brain area that is part of the limbic system, has a large number of dopamine-containing neurons, and plays a role in the learning and performing of certain behaviors in response to incentive stimuli (i.e., motivation and reinforcement).

Paranoia: A mental disorder characterized by the presence of systematized delusions, often of a persecutory character, involving being followed, poisoned, or harmed by other means, in an otherwise intact personality.

Physical dependence: An adaptive physiological state that occurs with regular drug use and results in a withdrawal syndrome when drug use stops.

Psychosis: A mental and behavioral disorder characterized by symptoms such as delusions or hallucinations that indicate an impaired conception of reality.

Psychosocial intervention: An individual or group interaction that examines both psychological and social aspects of a person's life (e.g., age, education, marital, and related aspects of a person's life history).

Rhabdomyolysis: An acute, potentially fatal disease of skeletal muscle characterized by muscle pain, weakness, and the production of red-brown urine.

Rush: A surge of euphoric pleasure that rapidly follows administration of a drug.

Seizure: Manifestation of a sudden onset of an abnormal mental or physical state, often characterized by complex behaviors, impaired consciousness, and convulsions.

Serotonin: A neurotransmitter that has been implicated in states of consciousness, mood, depression, and anxiety.

Serotonergic: Serotonin-mediated.

Stereotyped behaviors: Frequent, almost mechanical repetition of the same posture, meaningless gestures or movement, or form of speech (as in schizophrenia).

Substantia nigra: A discrete brain area that is part of the nigrostriatal system, interacts with the limbic system, has a large number of dopamine-containing cells, and is involved in learning to automatically execute complex movements triggered by a voluntary command; degenerative impairments in this area cause motor disturbances that occur in Parkinson's disease.

Synapse: A microscopic gap, cleft, or junction between neurons across which chemical signals (neurotransmitters) are transmitted.

Systolic blood pressure. The pressure exerted by the blood on the cavities of the heart at the moment when they contract.

Tachycardia: Rapid heartbeat, with or without arrhythmia and chest pain.

Tolerance: A condition in which higher doses of a drug are required to produce the same effect as experienced initially; often leads to physical dependence.

Toxic: Temporary or permanent drug effects that are detrimental to the function or structure of a cell, organ, or organ system.

Urticaria: An eruption of itching wheals, usually of systemic origin, which may be due to a state of hypersensitivity to food, drugs, or physical agents, such as heat or cold.

Ventral tegmental area: A discrete brain area that is part of the mesocortical system, interacts with the limbic system, has a large number of dopamine-containing neurons, and is involved in attention span and short-term memory.

Withdrawal: A psychological and/or physical syndrome caused by the abrupt cessation of the use of a drug in an habituated individual.

Appendix E
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The TIPs Series

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Other TIPs may be ordered by contacting the National Clearinghouse for Alcohol and Drug Information (NCADI), (800) 729-6686 or (301) 468-2600; TDD (for hearing impaired), (800) 487-4889.

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